



Public Health Assessment for

**FLAT CREEK IMM SITE
(a/k/a SUPERIOR WASTE ROCK)
SUPERIOR, MINERAL COUNTY, MONTANA
EPA FACILITY ID: MT0012694970
JUNE 30, 2009**

For Public Comment

**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE**
Agency for Toxic Substances and Disease Registry

Comment Period Ends:

AUGUST 14, 2009

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Prepared by:

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List of Abbreviations

AOEC	Association of Occupational and Environmental Clinic
ATSDR	Agency for Toxic Substances and Disease Registry
bgs	below ground surface
BLL	blood lead level
CDC	Centers for Disease Control and Prevention
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CREG	cancer risk evaluation guide
CRQL	Contract Required Quantitation Limit
CV	comparison value
DHHS	Department of Health and Human Services
EMEG	environmental media evaluation guide
EPA	U.S. Environmental Protection Agency
FDA	U.S. Food and Drug Administration
IARC	International Agency for Research on Cancer
kg	kilogram
IEUBK	Integrated Exposure Uptake Biokinetic Model for Lead in Children
IMM	Iron Mountain Mine and Mill
IPCS	International Programme on Chemical Safety
L/day	liters per day
LOAEL	lowest-observed-adverse-effect-level
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MCCCP	Montana Comprehensive Cancer Control Program
MCEHP	Mineral County Environmental Health and Planning
MCTP	Montana Central Tumor Registry
MDEQ	Montana Department of Environmental Quality
MDPHHS	Montana Department of Public Health and Human Services
mg/day	milligrams per day
mg/kg	milligrams per kilogram
mg/kg/day	milligrams per kilogram per day
mg/L	milligrams per liter
mL/hr	milliliters per hour
MRL	minimal risk level
NAS	National Academy of Science
NCDC	National Climatic Data Center
NOAEL	no-observed-adverse-effect-level
NOEL	no-observed-effect level
NPL	National Priorities List
NTCRA	non-time-critical removal action
ppb	parts per billion
ppm	parts per million
PWS	public water supply
QA/QC	quality assurance/quality control
RBC	risk-based concentration
RDA	recommended dietary allowance

RDI	reference daily intake
RfD	reference dose
RM	river mile
RMEG	reference dose media evaluation guide
SSL	soil screening level
STORET	STORage and RETrieval
TCLP	Toxicity Characteristic Leaching Procedures
TCRA	time-critical removal action
µg/day	micrograms per day
µg/dL	micrograms per deciliter
µg/L	micrograms per liter
USDA	U.S. Department of Agriculture
USGS	U.S. Geological Survey
WHO	World Health Organization
XRF	X-ray fluorescence spectrometer

1 Summary

In February 2004, the Agency for Toxic Substances and Disease Registry (ATSDR) received a petition for a public health evaluation of heavy metal contamination in soil and water in Superior, Montana. Residents expressed concern regarding exposures to waste tailings from the Iron Mountain Mine and Mill (IMM) site. Waste tailings from this mine site are washed down along the banks and floodplain of Flat Creek. Additionally, some waste tailings were used as fill material in the town. To address community concerns, ATSDR evaluated available data to determine whether harmful health effects are expected from exposures to heavy metal contamination in soil and water.

Overall, ATSDR concludes that coming into frequent contact with waste tailings on the Iron Mountain Mine and Mill site, the Flat Creek floodplain, and the town of Superior could harm people's health. Therefore, frequent, contact-intense activities¹ with the waste tailings may result in exposures that are a public health hazard. ATSDR reached the following pathway-specific conclusions.

- Waste tailings deposits, and sediment affected by waste tailings, on the Iron Mountain Mine and Mill property and Flat Creek floodplain area currently show arsenic and lead levels that are of public health concern if residents, particularly children, visit these areas and engage in contact-intense activities on a daily basis.
- Prior to removal actions, levels of arsenic and lead in soil for some town properties were of public health concern for children and adults who lived at or visited these areas and engaged in contact-intense activities on a daily basis.
- Currently, soil arsenic levels from two residential yards and soil lead levels from these two yards and two additional residential yards are of public health concern for children who live at or visit these four residences and engage in contact-intense activities on a daily basis.
- Heavy metals, including arsenic and lead, detected in soil at most residential and non-residential areas are currently not at levels of health concern for children and adults because the estimated chemical levels are below harmful effect levels. However, the potential exists for additional properties in town that were not sampled to contain waste tailings material with elevated levels of arsenic and lead and there is some uncertainty about which properties were actually remediated.
- Harmful health effects are not expected for children and adults following dermal contact with and incidental ingestion of Flat Creek surface water while wading and fishing downstream of the mine site.
- Except for lead, the limited data available suggest harmful health effects are not expected for resident's who use creek water for activities such as drinking, showering, bathing, cooking, and washing dishes. However, ATSDR's evaluation indicates that the potential exists for children who ingest 1 liter of Flat Creek water per day to have blood lead levels

¹ Contact-intense activities include digging with shovels and other tools, and playing with toys (like toy trucks and action figures) on the ground surface. Adults and children can be exposed by putting soiled hands or toys in their mouth or by breathing or eating dust generated by their activities.

of health concern. Furthermore, average levels of antimony and lead exceed regulatory guidelines and ATSDR considers efforts to reduce drinking water exposures prudent public health practice when chemical levels are above regulatory guidelines.

- Surface water in the Hall Gulch area would be at levels of public health concern if the water were to be frequently ingested. However, ATSDR would not expect people to drink, or wade in, this shallow, reddish-brown, surface water.
- Chemicals detected in groundwater samples from the three Superior public water supply wells, Flat Creek Spring, and a private well are not at levels of public health concern. However, in the past antimony levels in Flat Creek Spring and the private well exceeded regulatory guidelines. ATSDR considers efforts to reduce drinking water exposures prudent public health practice when chemical levels are above regulatory guidelines.
- ATSDR cannot evaluate groundwater exposures for residents on the north side of town who rely on private wells to obtain household water because few details are documented about these wells. With the exception of the one private well mentioned previously, no environmental data are available for these wells.

Based on ATSDR's conclusions, the agency makes the following recommendations.

1. Waste tailings-contaminated areas at the mine site and floodplain area should continue to be posted with warning signs advising the public that the soil contains arsenic and lead, which may pose a risk to public health.
2. Remedial actions should be considered to minimize exposure to waste tailings contamination, such as removal of waste tailings deposits on the mine site and floodplain area.
3. Additional characterization of town soil should be conducted to confirm the success or extent of the 2002 time-critical removal action.
4. Additional characterization of the four residential yards should be conducted to determine whether the detected soil arsenic and lead levels are truly representative of each yard.
5. Additional efforts, such as another mail-out to town residents or a town hall meeting, should be conducted to determine which areas of town need further characterization.
6. Hall Gulch should continue to be posted with warning signs informing the public that water coming out of the adit² has levels of metals that may pose a health risk.
7. Efforts to reduce drinking water exposures from Flat Creek intakes should be made because antimony and lead levels in the creek water are above regulatory guidelines.
8. Flat Creek Spring should be tested before it is used as an emergency public water supply to ensure antimony levels are below regulatory guidelines.
9. Private wells on the north side of town should be tested if it is determined they draw water from the shallow aquifer.

² Adits are the lowest level in a mine where water can drain out naturally.

10. Residents should be made aware of prudent public health measures they can take to reduce exposures and to protect themselves, their families, and visitors (see Appendix D).

In April 2009, the U.S. Environmental Protection Agency (EPA) listed the Flat Creek IMM site on its National Priorities List (NPL), and the first phase of residential soil sampling is expected to occur in summer 2009. ATSDR staff plan to work closely with EPA to maximize ATSDR's involvement in the early stages of the NPL listing.

2 Background

This public health assessment focuses on the Flat Creek IMM site, which includes the Iron Mountain Mine and Mill site, the Flat Creek floodplain, and the town of Superior. Figure 1, Appendix A, shows the location of the mine site in relation to the town of Superior. In this section, ATSDR provides background information on the mine site, Flat Creek, and the town of Superior.

2.1 Iron Mountain Mine and Mill Site

The Iron Mountain Mine and Mill site is located 3.5 miles northeast of the town of Superior. Discovered in 1888, the Iron Mountain mine was a zinc, lead, copper, silver, and gold mine [MDEQ 2004a]. This mine was the primary producer of silver, zinc, and lead in the area [USDA 2004]. Throughout its years of operation, the Iron Mountain Mine and Mill was responsible for bringing mineral deposits to the surface. These deposits were concentrated in soil and waste tailings at and near the mine site, and some were intentionally transported off site.

The Iron Mountain Mining Company operated the mine from 1888 until 1897. The owners of the land erected the mine along Flat Creek and built the mill 1 mile away. Large-scale operations began in 1891 when the Northern Pacific Railroad built a railroad branch line through Superior. In 1897, the mine closed for safety violations.

However, in 1905, the Iron Mountain Tunnel Company dug a new tunnel to reach a lower lode. From 1909 to 1953, the mine produced 7,535,084 pounds of zinc, 5,385,741 pounds of lead, 5,274 pounds of copper, 389,355 fine ounces of silver, and 19 fine ounces of gold. Several smaller mines in this area also produced additional amounts of these metals [MDEQ 2004a].

The mine changed ownership multiple times until its closure in 1954. The property is currently owned by ASARCO, a subsidiary of Grupo México. All that remains of the mill and other mining buildings are their foundations. A large waste rock pile and some waste tailings deposits still exist on the mine property, although a majority of the waste tailings have been washed downstream onto the Flat Creek floodplain [EPA 2002a].

2.2 Flat Creek

Flat Creek is about 9 miles long and flows in a southwesterly direction towards the town of Superior [USDA 2004]. The Iron Mountain Mine and Mill site is located along Flat Creek near its confluence with Hall Gulch. Shortly after entering the city limits, Flat Creek runs through a culvert before joining the Clark Fork River. Flat Creek is listed as a perennial stream, although in November 2003, a 200-foot section of the creek ran dry [USDA 2004]. Most of the length of Flat Creek lies within the jurisdiction of the U.S. Department of Agriculture (USDA) Forest Service, with access to the creek provided by a gravel road running roughly parallel to the creek.

The extent of the waste tailings deposits are highly variable from the mouth of Flat Creek [river mile (RM) 0.0], which is in the town of Superior, to its confluence with Hall Gulch (about RM 3.7), which is on the mine site. In areas not impacted by tailing deposits, the riparian vegetation³ is a dense, mature, lodgepole pine forest with deciduous trees and shrubs [USDA 2004]. However, the vegetation is not well established in some areas and active erosion of waste tailings

³ River banks are riparian areas, and the plants that grow there are called riparian vegetation. Riparian vegetation is extremely important because of the many functions it serves, such as river bank stabilization.

into Flat Creek was observed at waste tailings deposits along the creek, such as those found from RM 1.5 to RM 1.75 [USDA 2004].

2.3 The Town of Superior

The town of Superior is located in Mineral County, Montana, within the western thrust belt of the northern Rocky Mountains. The Clark Fork River flows to the west-northwest through town, dividing it into the south side, home to most residents and public facilities, and the north side, which has a smaller number of residents.

2.3.1 Land Use

The town of Superior is comprised largely of residential properties and service industries. There are approximately 410 homes in town. The economy for the area of Superior is based mostly on tourism and recreation related service industries [MDEQ 2003]. Hunting, camping, and other outdoor activities are common in the region.

2.3.2 Demographics

According to the 2000 census, approximately 900 persons live in the town of Superior [Bureau of the Census 2001]. Approximately 17% are age 65 and older and 9% are children 6 years or younger. Children are more susceptible than adults to exposure to hazardous substances because of their activity habits, small size, and developing bodies. Figure 2, Appendix A, provides additional demographic statistics.

2.3.3 General Climate

The town of Superior is located at an elevation of 2,710 feet [NCDC 2006]. The region is characterized by a cool, dry climate and wide seasonal and daily variations in temperature [USDA 2004]. The National Climatic Data Center (NCDC) reports an average annual precipitation of 16.58 inches and an average temperature range of 33.7°F in December to 86.8°F in August [NCDC 2006].

2.3.4 Hydrogeology

The town of Superior lies atop several layers of sediments associated with the Clark Fork valley and these sediments contain several water-bearing zones. The exact hydrogeology of the area is not well understood, but it is generally believed that water-bearing zones from the surface down to approximately 85 feet below ground surface (bgs) represent an unconfined aquifer that mixes freely with surface water (i.e., rivers and streams). Below about 85 feet bgs, water-bearing zones are believed to be confined by less permeable layers of fine-grained sediments. In general, groundwater in the area around Superior flows from the mountains to the valley, and within the valley it flows mainly to the north-northwest, roughly parallel to surface water flow in the Clark Fork River [MDEQ 2003].

2.3.5 Groundwater Use

Since the early 1900s, the majority of town residents have been connected to the public water supply (PWS). Previously, the PWS source for the town of Superior was a spring adjacent to Flat Creek. However, the Mountain Water Company (former PWS owner) discontinued use of Flat Creek Spring in 1997 when antimony was detected at concentrations above the U.S. Environmental Protection Agency's (EPA's) maximum contaminant level (MCL) [EPA 2001b,

MDEQ 2004b]. Currently, the spring is not in use, but it is maintained as an emergency drinking water source [URS 2001]. Although named “Flat Creek Spring”, the spring surfaces at a higher elevation than Flat Creek [EPA 2001b]. As a “gravity flow spring”, it arises from area groundwater [MDEQ 2004b].

Ownership of the PWS was transferred from the Mountain Water Company to the town of Superior in October 2000. The current PWS has a total of 430 connections. There are three production wells for this system: Well 1, Well 2, and Well 3. All three wells are located within the city limits of the town of Superior and drilled into the confined aquifer at depths of 105.5 feet bgs (Well 1), 118 feet bgs (Well 2), and 214 feet bgs (Well 3). Each well has its own treatment plant. The town of Superior tests these wells for water quality in accordance with federal standards. Figure 1, Appendix A, shows the locations of these wells.

Most residents living in the town of Superior receive drinking water from the PWS, but a few homes on the north side of town obtain water from private wells. In general, these private wells draw water from the deep aquifer (more than 85 feet bgs), which is believed to be confined. However, several homes do have wells that draw water from less than 85 feet bgs [MDEQ 2003]. It is not known whether these wells are currently used as a drinking water source.

There is also one residence located north of town limits that is not served by the PWS. This family draws drinking water from two distinct sources—a private groundwater-fed well and a tap diverted from Flat Creek, approximately 2 miles south of the Iron Mountain Mine and Mill site [EPA 2002a].

2.4 Remedial Activities

In 1993, the Montana Department of State Lands, Abandoned Mine Reclamation Bureau conducted an investigation of potential health and environmental risks associated with the Iron Mountain Mine and Mill site [EPA 2002d]. This assessment found that multiple metals, including arsenic, copper, mercury, lead, zinc, cadmium, manganese, and antimony, were present in soils in concentrations more than three times background levels [EPA 2002d].

In 1998, ASARCO conducted reclamation activities that included re-vegetation of some Flat Creek tailings and removal of other tailings from Flat Creek to an impoundment on ASARCO property [EPA 2002d]. However, there are no post-remediation sampling data available to confirm the success or extent of these remediation activities [EPA 2002d].

In January 2002, MDEQ requested that EPA evaluate the town of Superior for a possible removal action [EPA 2002c]. In June 2002, EPA conducted additional surface and subsurface soil sampling and found multiple areas of Superior with soil containing elevated levels of arsenic and lead [EPA 2002d]. Also in 2002, Mineral County Environmental Health and Planning (MCEHP) collected blood lead and urine arsenic samples from individuals living in Superior [MCEHP 2002c]. Section 4.4 contains the results of the blood lead and urine arsenic sampling event.

In August 2002, EPA began a time-critical removal action (TCRA) in Superior [EPA 2002c]. First, contaminated soils at the high school track, the county fairground, along the fence line of a residential property, and the driveway of a residential property were to be excavated [EPA 2002e]. A sample from each of these four locations was run for Toxicity Characteristic Leaching Procedures (TCLP) and all four samples failed TCLP for lead. Contaminated soils and waste tailings located in other areas, including rights-of-way and residential properties, were to be

included in the removal action if the average surface soil concentration exceeded 400 parts per million (ppm) arsenic or 3,000 ppm lead, or both. Overall, EPA removed approximately 6,500 cubic yards of contaminated tailings and soil from town locations [EPA 2003a].

With regard to the time-critical removal, contaminated soil was removed to a depth of 12 inches, except in gardens where it was removed to 24 inches. Excavated areas were backfilled using clean soil and top soil, other materials comparable with existing materials, or a combination of cleaned, compacted gravel and asphalt [EPA 2002c]. Limited soil data from the excavation effort are available. The contaminated soil was transported to the county airport where Environmental Restoration Inc. and Envirocon of Missoula treated it with Portland cement and installed liners to minimize leaching.

Although inactive, the mine site can be reached by a gravel road (USFS Road #194) that roughly parallels Flat Creek [USDA 2004]. Waste tailings are still present at the mine site and in variable deposits in the floodplain of Flat Creek downstream from the mine site [USDA 2004]. Site investigations indicate that tailings are continuously mobilizing into Flat Creek by runoff and by erosion augmented by wildfire damage and post-precipitation flooding [EPA 2001a, EPA 2001b, USDA 2004]. Assessment of Flat Creek indicates that tailings are present for up to several miles south of the mine site, ranging from trace amounts in the steeper stream segments to extensive deposits in the flatter and more open floodplain [USDA 2004]. Additionally, both the Montana Department of Environmental Quality (MDEQ) and the USDA Forest Service have documented metals in Flat Creek surface water [EPA 2001a, MDEQ 2004b].

MDEQ listed the Iron Mountain Mine and Mill site on its Comprehensive Environmental Cleanup and Responsibility Act Priority List in 2004, and indicated that this is a high-priority site [MDEQ 2004b]. In April 2009, EPA listed the Flat Creek IMM site on its National Priorities List (NPL).

2.5 ATSDR Activities

In 2002, ATSDR became involved with the Iron Mountain and Mill site. A town of Superior resident contacted ATSDR to request that the agency initiate communication with Mineral County. In response, ATSDR worked with the EPA, the Montana Department of Public Health and Human Services (MDPHHS), and the MCEHP to create a fact sheet for the community. ATSDR reviewed soil and water sample results from Flat Creek, performed a site visit of the area, and communicated with local residents.

Since 2002, ATSDR has received several letters from concerned community members with questions about exposures to heavy metals and other contaminants associated with the Iron Mountain Mine and Mill site, the Flat Creek floodplain, and the town of Superior. Section 7 of this document, Community Health Concerns, addresses the health concerns expressed by local residents.

Most recently, in September 2008, ATSDR staff conducted a site visit. The purpose of the visit was to meet with ATSDR's petitioner to discuss the site. ATSDR staff also met with county staff and toured the site and possible contamination source areas (visible waste tailings along Flat Creek and on the mine property) [ATSDR 2008a].

3 Exposure Pathway Evaluation

To determine whether people are being exposed to contaminants or whether they were exposed in the past or will be exposed in the future, ATSDR examines the path between a contaminant and a person or group of people who could be exposed. Completed exposure pathways have five required elements. ATSDR evaluates each pathway at a site to determine whether all five factors exist and people are being exposed, were exposed, or may be exposed in the future. These five factors or elements must exist for a person to be exposed to a contaminant:

- (1) a source of contamination
- (2) transport through an environmental medium
- (3) a point of exposure
- (4) a route of human exposure, and
- (5) an exposed population.

ATSDR classifies exposure pathways in one of the following three categories.

- *Completed Exposure Pathway.* ATSDR calls a pathway “complete” if it is certain that people are exposed (or were exposed or will be exposed) to contaminated media. Completed pathways require that the five elements exist and indicate that exposure to the contaminant has occurred, is occurring, or will occur.
- *Potential Exposure Pathway.* Potential pathways are those in which at least one of the five elements is missing, but could exist. Potential pathways indicate that exposure to a contaminant could have occurred, could be occurring, or could occur in the future.
- *Eliminated Exposure Pathway.* In an eliminated exposure pathway, at least one of the five elements is missing. From a human health perspective, pathways can be eliminated from further consideration if ATSDR is able to show that (1) an environmental medium is not contaminated or that (2) no one is exposed to contaminated media.

In the following text, ATSDR describes the exposure pathways associated with the Flat Creek IMM site. Also, completed exposure pathways are summarized in Table 1, Appendix B; potential and eliminated exposure pathways are summarized in Table 2, Appendix B.

3.1 Soil and Sediment

Exposure to soil and sediment occurs primarily through dermal contact. In addition, people might accidentally ingest soil and exposed sediment, as well as dust that is generated from disturbing the soil and sediment. Preschool age children tend to swallow more soil and exposed sediment than do any other age group because they have more contact with soil and sediment through their play activities and they tend to exhibit mouthing of objects. Children in elementary school, teenagers, and adults tend to swallow much smaller amounts of soil and sediment. The amount of grass cover in an area, the amount of time spent outdoors, and weather conditions also influence people’s exposure to soil.

3.1.1 Iron Mountain Mine and Mill Site and Flat Creek Floodplain

Although reclamation activities have occurred, including the re-vegetation of some Flat Creek tailings, waste tailings still exist on the mine site and along portions of Flat Creek. Site investigations indicate that erosion augmented by wildfire damage and precipitation runoff into Flat Creek have mobilized waste tailings such that they extend into the soil and the streambed sediment [EPA 2001a, EPA 2001b, USDA 2004]. Soil, sediment and waste tailings downstream from the Iron Mountain Mine and Mill co-mingle in the Flat Creek floodplain area. For the purpose of its health evaluation, ATSDR addresses waste tailings under the designation of “soil and sediment” because the exposure scenario is similar (i.e., dermal contact and incidental ingestion).

A past, current, and future completed exposure pathway to surface soil, sediment, and waste tailings exist for people engaged in hunting, fishing, wading, hiking, and other recreational activities on the Iron Mountain Mine and Mill site and along the Flat Creek floodplain. ATSDR staff noted broken bottles and plastic containers on the mine site during their 2008 site visit [ATSDR 2008a]. However, it is not known how frequently recreational activities occur in areas where contamination exists.

Of note, in February 2002, MCEHP released two environmental health notices to the public to raise awareness that preventative measures should be taken to reduce exposure to lead and arsenic in contaminated soils in the Flat Creek area [MCEHP 2002a, MCEHP 2002b]. In addition, portions of the mine site and Flat Creek are posted with warning signs advising the public that the soil may be contaminated with arsenic and lead, which may pose a risk to public health [MCEHP 2002b, MCEHP 2002c]. The county sanitarian stated the area is used only sporadically for recreational activities [T. Read, MCEHP, personal communication, May 18, 2006]. Although the area is used only sporadically for recreational activities, the agency supports these measures to protect public health in areas of known contamination.

3.1.2 The Town of Superior

In the past, waste tailings from the mine were used as surface soil fill on public and residential properties [EPA 2001a, EPA 2002a]. Contaminated areas were driveways, yards, gardens, public rights-of-way (e.g., along roads), public schools and the county fairgrounds. Of note, all residences, whether located *within* town limits or *just north* of town limits, are considered “the town of Superior” for this public health evaluation because the exposure pathway elements for each residence are similar.

Before the 2002 TCRA, a completed exposure pathway existed to waste tailings used as fill in the town of Superior. At the Superior High School track, athletes and other school children came in contact with waste tailings during play and athletic activities. At private residences where mine tailings were used for driveway fill and for yards and gardens, residents were exposed to surface soil when it was stirred up by gardening, moving vehicles, and children playing. In addition, residents were exposed to surface soil contaminated with waste tailings when visiting high traffic areas within town, including the county fairgrounds.

Because only limited data are available to confirm the success or extent of the 2002 TCRA, a current and future completed exposure pathway to soil exists within the town of Superior. Local residents and area visitors could be exposed to soil contaminated with waste tailings within the town of Superior, including areas that were not targeted for removal actions.

3.2 Surface Water

Exposure to surface water occurs through dermal contact and incidental ingestion during activities such as swimming, wading, and fishing. In some instances, surface water is used for domestic supply, which leads to surface water exposures through drinking, showering, bathing, dishwashing, and other household activities.

As stated previously, waste tailings from the mine site can enter into Flat Creek. A past, current, and future completed exposure pathway to surface water exists for recreational users of Flat Creek. In general, because the creek is not especially deep, ATSDR assumes wading and fishing would be the primary recreational activities, not necessarily swimming. ATSDR expects, given the climate in Mineral County, that wading is limited to warmer months [NCDC 2006]. The fishing season for rivers and streams in western Montana lasts about 6 months [MFWP 2006]. Residents who occasionally wade or fish in the creek are exposed to the creek water through dermal contact and incidental ingestion.

In addition, one family obtains part of their household water supply from a Flat Creek intake [EPA 2002a]. For this family, a past, current, and future completed exposure pathway exists through activities such as drinking, showering, bathing, cooking, and washing dishes with the surface water.

Also of note, the old mine shaft located in Hall Gulch discharges water that flows over a waste rock pile on the mine site. The surface water discharging from this mine adit is very shallow water (1 inch) and has a reddish-brown color. The water pools in two areas. Although there is evidence of trespassing in Hall Gulch, ATSDR would not expect people to drink, or wade in, this shallow, reddish-brown, surface water. Furthermore, Hall Gulch is posted with a warning sign to ensure the public is aware that water coming out of the adit has levels of metals that may pose a health risk [MCEHP 2002b, MCEHP 2002c]. At this time, ATSDR consider Hall Gulch surface water an eliminated exposure pathway.

3.3 Groundwater

Exposure to groundwater occurs when it is pumped to the surface to be used for domestic supply. Dermal contact and ingestion are the primary routes of exposure to groundwater contaminated with heavy metals. Groundwater exposures occur through drinking, showering, bathing, dishwashing, and other household activities.

3.3.1 *Town of Superior Wells*

Most residents receive water from the PWS, which currently draws water from three wells located within the city limits [MDEQ 2003]. Water from the town wells is treated before entering the distribution system. For the purpose of this health consultation, ATSDR considers past, current and future use of the three town of Superior wells a completed exposure pathway.

3.3.2 *Flat Creek Spring*

Prior to 1998, Flat Creek Spring was the primary PWS for the town of Superior. Therefore, Flat Creek Spring represents a past completed exposure pathway. Following detection of elevated levels of antimony, this water source was shut down.

However, the spring is maintained for use as an emergency drinking water source. Because the spring is not a primary source of drinking water, the spring currently represents an eliminated

exposure pathway. However, future use of the spring represents a potential exposure pathway because the spring could be used during emergencies.

3.3.3 *Private Wells*

Several residents on the north side of town rely on private wells to obtain household water. However, limited details are available about these wells. Although the majority of private wells appear to be deeper than 85 feet bgs, a few are believed to draw water from 85 feet bgs or shallower [MDEQ 2003]. Private wells represent a past, current and future completed exposure pathway.

4 **Environmental and Biological Data**

ATSDR reviewed data collected from several sampling events. These data are assumed to represent contamination levels at the site. Of note, however, this data review was not intended to be an exhaustive compilation of all data ever collected at the site. ATSDR reviews data relevant to the identified exposure pathways and community concerns.

As an initial screen, ATSDR reviewed the environmental soil, sediment, waste tailings, surface water, and groundwater data that were available to the agency to determine whether the site's maximum detected chemical concentrations are above each chemical's protective health-based comparison values (CVs). ATSDR also reviewed available biological data.

A health-based CV is an estimate of daily human exposure to a chemical that is not likely to result in harmful health effects over a specified duration of exposure. ATSDR CVs are developed for specific media (air, water, and soil) and for specific durations of exposure (acute, intermediate, and chronic). This initial screen also identifies those chemicals with no CVs.

Comparison values and health guidelines used by ATSDR scientists include ATSDR's cancer risk evaluation guides (CREGs) and environmental media evaluation guides (EMEGs). If an ATSDR CV is not available for a particular chemical, ATSDR can also screen environmental data with CVs developed by other sources, including EPA's risk-based concentrations (RBCs). These CVs and health guidelines, as well as all other health-based screening criteria, represent conservative levels of safety; they are not thresholds of toxicity. Although concentrations at or below a CV may reasonably be considered safe, concentrations above a CV will not necessarily be harmful. To ensure that they will protect even the most sensitive populations (such as children or the elderly), CVs are intentionally designed to be much lower, usually by two or three orders of magnitude, than the corresponding no-observed-adverse-effect-levels (NOAELs) or lowest-observed-adverse-effect-levels (LOAELs) on which the CVs were based. Most NOAELs and LOAELs are established in laboratory animal studies; relatively fewer are derived from epidemiologic (chiefly occupational) studies. All ATSDR health-based CVs are non-enforceable and used for screening purposes only.

When determining what environmental guideline value to use, ATSDR follows a general hierarchy. Hierarchy 1 includes ATSDR environmental guidelines such as CREGs and chronic EMEGs. In the absence of these values, Hierarchy 2 values (including ATSDR's reference dose media evaluation guide, or RMEG), may be selected. When ATSDR environmental guidelines listed in the hierarchy are unavailable, those from other sources are considered [ATSDR 2005b].

ATSDR selects chemicals for further consideration if either (a) their maximum concentrations associated with an exposure pathway exceed a relevant CV, or (b) there are no CVs listed for them. The following text provides the results of ATSDR's screening analysis.

4.1 Soil and Sediment Data

Data from the Iron Mountain Mine and Mill and the Flat Creek floodplain area will be presented first, followed by data from the town of Superior. As stated previously, for the purpose of this health consultation, ATSDR is addressing waste tailings under the designation of "soil and sediment." Because CVs do not exist for sediment and waste tailings, ATSDR screened these data with available health-based CVs for the soil medium.

4.1.1 The Iron Mountain Mine and Mill and the Flat Creek Floodplain

Environmental sampling events occurred on the Iron Mountain Mine and Mill site and Flat Creek floodplain to assess contamination of these areas. No soil samples were collected from the mine site or floodplain area. Samples were collected directly from waste tailings material and from sediment affected by waste tailings. ATSDR compiled these data from several sources.

- EPA collected six sediment samples from the Iron Mountain Mine and Mill site at the mine adit and Flat Creek floodplain area between October 16–18, 2001, and analyzed the samples for 23 analytes [EPA 2002a]. All data were validated and are acceptable for use as qualified in the data validation report [EPA 2002a]. The metals antimony, arsenic, cadmium, iron, lead, manganese, thallium, and zinc were detected in the sediment samples at levels exceeding soil CVs. All other analytes were below their respective health-based soil CVs. Table 3, Appendix B, provides results for those analytes that were detected above their respective CVs.
- As part of its 2002 site investigation, the USDA Forest Service collected sediment and waste tailings samples from the Flat Creek streambed [USDA 2004]. Because typical recreational users are expected to be exposed to surface materials, ATSDR included in its review the sediment and waste tailings samples collected from less than 12 inches bgs. The USDA Forest Service tested the samples for seven metals. The data analyses and reporting appear to have been conducted in accordance with standard data quality practices, but no documentation was available to confirm the level of quality assurance/quality control (QA/QC) [USDA 2004]. The metals antimony, arsenic, cadmium, lead, manganese, and zinc were detected at levels exceeding soil CVs in both sediment and waste tailings samples. Tables 3 and 4, Appendix B, provides results for those analytes that were detected above their respective CVs from the Flat Creek streambed.
- In addition to the data summarized in Tables 3 and 4, Appendix B, the USDA Forest Service's report summarized data from previous examinations of contaminants in Flat Creek streambed sediment and Flat Creek road soil [USDA 2004]. The streambed sediment samples in this study had concentrations of analytes that exceeded soil CVs, including antimony (range: 18.5–4,890 ppm), arsenic (range: 12–3,970 ppm) and lead (range: 89.3–27,900 ppm). The Flat Creek road soil samples contained concentrations of antimony (range: <9.1–3,070 ppm), arsenic (range: <5.7–1,080 ppm) and lead (range: 59.8–26,300 ppm). Because the USDA Forest Service's report did not state the number of

samples collected nor the depth of the samples, these data have not been included in Tables 3 and 4, Appendix B.

Of note, EPA and the USDA Forest Service detected several analytes in background samples such as upstream from the mine site, at the upper ASARCO boundary, and along the Clark Fork River. However, concentrations of all but one metal were below CVs. Arsenic levels ranged from 8.8–19.9 ppm, which is above its CVs but within background levels typically found in Montana [MDEQ 2005].

4.1.2 The Town of Superior

Environmental soil sampling events occurred in the town of Superior prior to the 2002 time-critical removal effort. In addition, limited soil samples were taken during the 2002 removal effort from properties that were excavated. Soil samples were also collected during excavation of a public water main line in 2007. The following text provides ATSDR's summary of the available soil data collected in the town of Superior.

4.1.2.1 Soil Levels Prior to Removal Activities

To determine contamination levels in the town of Superior, EPA conducted a focused assessment of soil prior to removal activities [EPA 2002a]. The sampling approach was based on the subjective selection of sampling locations in town where contamination was expected to occur [EPA 2002d]. EPA mailed town of Superior residents a letter, with an attached access consent form, letting them know that it planned to do sampling and to contact MCEHP if they had information about the location of waste tailings in the town [EPA 2002b].

In June 2002, a total of 635 soil samples were collected for X-ray fluorescence spectrometer (XRF) on-site screening from 66 residential properties, 20 right-of-way locations, 9 city/county and open space properties within and around Superior, and at the Superior High School track. Ten additional residential properties were originally identified for sampling, but were not sampled because of the distance from the town of Superior, later refusal of access, or change of ownership [EPA 2002d].

In general, for residential properties, a minimum of two composite surface soil samples were collected from each zone. A zone was considered a 5,000 square foot or smaller area. For right-of-ways, discrete surface samples were collected at 50-foot intervals on each side of the street. For open space samples, one composite sample was collected from each zone [EPA 2002d].

Soil samples were screened by XRF in the field for 24 to 26 inorganic constituents [EPA 2002a]. Most samples were surface soil (0-3 inches bgs). Several deeper soil samples (9-12 inches bgs) were collected in locations with elevated surface soil metal concentrations. The XRF field screening results for arsenic and lead are presented in Table 5, Appendix B. Only about 15% of the soil samples showed detectable levels of arsenic. However, it should be noted that the detection limit of the XRF screening technique for arsenic is relatively high and arsenic concentrations may be masked by high lead concentrations when using this method.

To confirm field XRF results, EPA analyzed 10% of the soil samples (64 samples) in the laboratory. Because the laboratory used lower detection limits and had less contaminant interference, the sample results did not contain as many non-detects. For example, arsenic was detected in 97% of the samples. Table 6, Appendix B, presents the laboratory analysis results for arsenic and lead. Table 7, Appendix B, presents the XRF and laboratory results for the other

analytes detected above CVs in town soils. In addition, four samples were submitted for TCLP analysis. All four samples failed TCLP for lead [EPA 2002e].

Of note, EPA conducted a preliminary analysis of town soil in 2001 [EPA 2002d]. The 2001 preliminary analysis sampled a smaller subset of town locations than the 2002 assessment. The 2001 smaller data set provides no additional insight to the nature and extent of town soil conditions beyond the results reported in the 2002 assessment; as such, the 2001 data are not included in this report.

However, the 2001 preliminary analysis included a single sample from a background location on the west side of town where no mine tailings have reportedly been transferred [EPA 2002d]. EPA tested for five analytes at this location: antimony, arsenic, cadmium, lead, and mercury. The only analyte to exceed its CV in this location was arsenic, but the reported concentration of 3.9 ppm was within background levels typically found in Montana [MDEQ 2005].

Overall, EPA reported concentrations of lead and arsenic in soil exceeding CVs at various town locations, including rights-of-way, private residences, public spaces, and the Superior High School track (Tables 5 and 6, Appendix B). Additional analytes were also observed at concentrations above CVs, including antimony, cadmium, iron, and manganese (Table 7, Appendix B). The maximum concentrations of these analytes were measured at the same locations where maximum values of arsenic and lead were detected.

4.1.2.2 Soil Levels Following Removal Activities

From August to November 2002, EPA conducted a focused, TCRA in the town of Superior. Contaminated soil was removed from public and private properties, including the high school track, the fairgrounds, and two residential locations [EPA 2002c, EPA 2003a]. In addition to these properties, requirements for removal action appear to include all locations with average surface soil concentrations above 400 ppm of arsenic or 3,000 ppm of lead [EPA 2002c]. In addition, the USDA Forest Service conducted its own removal at a home it owned.

However, the documentation received by ATSDR was not entirely clear regarding what areas of town underwent removal actions. For example, EPA's initial report [EPA 2002e] stated the addresses of two residences where removal actions would be occurring; however, a later sampling report [EPA 2002f] provided the addresses of three different residences where excavation samples were collected. These residences did not necessarily meet the removal action requirements of soil concentrations above 400 ppm of arsenic or 3,000 ppm of lead. This leaves some uncertainty about which properties were actually remediated and the levels of arsenic and lead that remain.

As stated previously, limited excavation sampling results were available to ATSDR from the removal effort [EPA 2002f]. The following text summarizes these data:

- For arsenic, three residential properties were sampled. Arsenic was found in these residential soil samples at levels of not detected, 45 ppm, and 110 ppm. The fairgrounds had arsenic levels of 51 ppm and 130 ppm. The high school track showed arsenic levels ranging from not detected to 220 ppm. Three samples from rights-of-way within the town did not detect arsenic.
- For lead, the three residential properties showed levels of 120 ppm, 230 ppm, and 560 ppm. The fairgrounds had lead levels of 240 ppm and 1,300 ppm. The high school track

showed lead levels from not detected to 420 ppm. Three samples from rights-of-way within the town indicated lead levels of 34 ppm, 55 ppm and 57 ppm.

Of note, these excavation data were marked “draft.” The available data did not include information like whether these excavation data represent XRF screening results or laboratory analysis results, whether these data represent surface soil or depth samples, or whether these data were discrete or composite samples. Because the data are limited, ATSDR finds that these excavation data might not accurately describe conditions in town following the removal effort. Additional sampling data are needed to confirm the success and extent of the 2002 removal activities.

Therefore, to *estimate* soil concentrations of arsenic and lead currently in the town, ATSDR attempted to compile a subset of data from the available 2002 soil data. Because the laboratory used lower detection limits, had less contaminant interference, and had less non-detects in the sample results compared to the XRF data, ATSDR compiled its subset of data from the laboratory samples (64 samples).

From the original 64 laboratory samples collected in 2002, those falling above cleanup criteria (400 ppm of arsenic or 3,000 ppm of lead), as well as those found at the same location as samples above cleanup criteria, were removed from the data set. At a minimum, locations with this level of contamination were assumed to have been subject to the removal action and therefore do not represent current conditions. In addition, ATSDR assumed the limited excavation sampling data were collected from locations where removal efforts occurred so these data were removed from the data set as well. The remaining subset of data, which is composed of 42 samples, represents ATSDR’s *estimate* of arsenic and lead soil concentrations currently in the town. Of note, this subset of 42 data results provides some perspective on soil contamination currently in Superior, but by no means is definitive. The following was found.

- For arsenic, 41 of the 42 soil samples contained measurable concentrations. For the 22 residential yards sampled, there were four yards with arsenic above ATSDR’s EMEG of 20 ppm. Arsenic concentrations for three of these four yards also exceeded the MDEQ Remediation Division’s generic action level of 40 ppm for arsenic in residential surface soil [MDEQ 2005]. Overall, the average arsenic concentration in these four yards was 155 ppm, with a maximum concentration of 362 ppm. For the remaining 20 non-residential samples (including rights-of way and open space locations), five samples were above ATSDR’s EMEG. The average arsenic concentration in these five samples was 57 ppm, with a maximum concentration of 92 ppm. For those samples with measurable arsenic concentrations (41 samples), all samples were above ATSDR’s CREG.
- For lead, all 42 soil samples contained measurable concentrations. For the 22 residential yards sampled, there were four yards with lead above EPA’s soil screening level (SSL) of 400 ppm. The average lead concentration in these four yards was 982 ppm, with a maximum concentration of 1,680 ppm. For the remaining 20 non-residential soil samples (including rights-of way and open space locations), four samples were above the lead SSL. The average lead concentration in these four samples was 561 ppm, with a maximum concentration of 689 ppm.

Of note, the residential yards, rights-of-way, and open space locations where lead exceeded the EPA SSL were the same locations where arsenic exceeded the ATSDR EMEG. Antimony exceeded the ATSDR RMEG in these same locations as well. For the subset of 42 data results,

the maximum level of antimony was detected at a residence (63.5 ppm antimony). Cadmium, iron, and manganese were all below their CVs in the subset of 42 samples.

In 2007, soil samples were collected during excavation of a public water main line and analyzed for arsenic and lead. Samples were collected mostly from a sample depth of 0-6 inches bgs from three areas: West Riverside and 6th Avenue, Diamond Road and Main Avenue, and Mullan Road. Levels of arsenic ranged from not detected to 81 mg/kg, with a large percentage of the samples (80%) not detecting arsenic. Levels of lead ranged from not detected to 804 mg/kg, with an average of 91 mg/kg. Only four of the 84 samples analyzed for lead were above EPA's SSL of 400 ppm, and three of those samples were found along Mullan Road. Like the excavation samples from the removal action, ATSDR does not know whether these data represent XRF screening results or laboratory analysis results, or whether the data collection and analysis followed QA/QC procedures.

4.2 Surface Water Data

Surface water sampling events occurred on the Iron Mountain Mine and Mill site and downstream Flat Creek floodplain area to assess surface water contamination. Table 8, Appendix B, contains the results of the surface water data ATSDR compiled from these sources.

- Between July 1996 and June 1998, surface water in the Flat Creek area was sampled at the request of ASARCO and MDEQ and analyzed for 14 inorganic constituents [MFG 2000]. No information is available for this data set regarding sampling technique or analysis method. From the limited information available, it appears 13 surface water samples were collected from near the mine adit and waste rock pile area and seven surface water samples were collected downstream of the mill site in Flat Creek.
- Between October 16 and 18, 2001, EPA collected six surface water samples at the Iron Mountain site and along Flat Creek downstream from the mine [EPA 2002a]. One of the six samples was collected from surface water directly below the waste pile found in Hall Gulch. EPA also collected three background surface water samples. EPA tested the samples for total metals and dissolved constituents (23 analytes). All data were validated and are acceptable for use as qualified in the data validation report [EPA 2002a].
- In 2002, the USDA Forest Service performed a site investigation at Iron Mountain [USDA 2004]. The USDA Forest Service collected 11 surface water samples at key points along Flat Creek, downstream from the mine. The USDA Forest Service also collected a single background sample at the upper ASARCO boundary, upstream from the mine. These samples were tested for seven analytes (total antimony, arsenic, cadmium, copper, iron, lead, and zinc). No documentation is available to confirm the level of data QA/QC [USDA 2004].

Overall, six analytes exceeded their corresponding drinking water CVs in surface water samples: antimony, arsenic, cadmium, lead, manganese, and zinc (see Table 8, Appendix B). The location of the maximum concentration of each metal was from the surface water in Hall Gulch (see Figure 1, Appendix A).

In addition, EPA collected one water sample from the private residence located north of town that draws drinking water from a tap diverted from Flat Creek [EPA 2002a]. The sampling data were validated and are acceptable for use as qualified in the data validation report [EPA 2002a].

Of the 23 analytes measured, antimony and thallium were detected above their respective CVs. The detected concentration of antimony of 12 parts per billion (ppb) was above its ATSDR child RMEG and above its EPA MCL; the concentration of thallium (0.62 ppb) was slightly above its EPA maximum contaminant level goal (MCLG) (0.5 ppb) but below its EPA MCL (2 ppb). Of note, although arsenic was not detected, the arsenic detection limit was equal to its MCL (10 ppb) and above ATSDR's CREG (0.02 ppb).

At Flat Creek background surface water locations upstream from where mining operations occurred, no analytes exceeded relevant CVs.

In addition to the data compiled in Table 8, Appendix B, the USDA Forest Service cites two previous reports that found antimony in surface water at levels ranging from 1 to 42 ppb [USDA 2004]. These maximum values are above ATSDR's child RMEG of 4 ppb for drinking water.

4.3 Groundwater Data

This section summarizes available groundwater sampling data for the public water system (PWS) and a private well.

4.3.1 Public Water System

Groundwater sampling data are available for the distribution system, Well 1, Well 2, Well 3, and Flat Creek Spring [EPA 2002a, MDEQ 2000, MDEQ 2007]. Table 9, Appendix B, contains the results of the PWS groundwater data.

Although the source documents are unclear, it is ATSDR's understanding that "distribution system" data reflect finished drinking water (i.e., treated water). It is also ATSDR's understanding that Wells 1, 2, and 3 data reflect unfinished groundwater (i.e., water before treatment). No documentation was available to confirm the level of QA/QC. The data indicate the following.

- For the distribution system, antimony exceeded ATSDR's child RMEG of 4 ppb and EPA's MCL of 6 ppb in 1 of 43 samples. Although below EPA's MCLs, arsenic exceeded ATSDR's CREG of 0.02 ppb in 1 of 3 samples and cadmium exceeded ATSDR's chronic child EMEG in 1 of 3 samples.
- For Wells 1, 2, and 3, antimony exceeded ATSDR's child RMEG and EPA's MCL in 1 of 38 samples. Although below EPA's MCL, arsenic exceeded ATSDR's CREG in 6 of 26 samples and ATSDR's chronic child EMEG in 1 of 26 samples.

As stated previously, Flat Creek Spring arises from area groundwater [MDEQ 2004b]. The town of Superior sampled water from the spring between 1993 and 1998, during which time it served as the primary source for the public water supply. Once in 1993 and once in 1997, the spring water was analyzed for cadmium but it was not detected on either occasion. Once in 1997, the spring water was analyzed for lead but it was not detected. Flat Creek Spring water was analyzed for arsenic on five occasions. Arsenic was detected on three occasions at a level of 1 ppb, which exceeds ATSDR's CREG but is below EPA's MCL. Antimony was detected in 20 of 33 spring water samples at levels ranging from 2–34 ppb. Nineteen of the 20 antimony detections exceeded both ATSDR's child RMEG and EPA's MCL. As stated previously, this water source has been on standby since 1997 but can serve as an emergency drinking water source, if needed.

The spring was also tested after it served as the primary source for the public water supply. In September 2000, MDEQ collected a single sample from the spring at the point where it discharges through a pipe into Flat Creek [MDEQ 2000]. Five metals were measured: antimony, arsenic, lead, mercury, and zinc. In 2001, EPA sampled the spring and tested for total metals [EPA 2002a]. No documentation is available to confirm the level of data QA/QC. Only antimony, at 29 ppb, was detected above CVs [EPA 2002a].

4.3.2 Private Groundwater Wells

One private residence located north of town draws drinking water from a private groundwater-fed well [EPA 2002a]. In October 2001, EPA collected a sample at this location and tested it for 23 inorganic constituents. All data were validated and are acceptable for use as qualified in the data validation report [EPA 2002a]. Of the 23 analytes measured, only antimony and thallium were detected at or above CVs. The detected concentration of antimony (24 ppb) was above its ATSDR child RMEG and above its EPA MCL. The concentration of thallium (0.52 ppb) was equivalent to its EPA MCLG and below its EPA MCL. Of note, the arsenic detection limit was at or below EPA's MCL, but above ATSDR's chronic child EMEG and CREG values.

As stated previously, several residents on the north side of town rely on private wells to obtain household water. However, few details are documented about these wells and no environmental data are available to evaluate.

4.4 Biological Data

To evaluate exposure in people, arsenic can be measured in urine and lead can be measured in blood. However, biological sampling has limitations. Sampling methods must be used judiciously and applied appropriately to successfully understand site-specific environmental exposures.

During February 2002, Mineral County Public Health and the MDPHHS offered urine arsenic tests and blood lead screening to residents of Superior [MCEHP 2002c]. The following text provides general information about urine arsenic and blood lead biological sampling methods as well as Superior resident's results.

4.4.1 Urine Arsenic

To evaluate exposure in humans, arsenic can be measured in urine, hair, nails or blood. However, urine arsenic is the most reliable method for measuring arsenic exposure, particularly exposures that occurred within a few days of the specimen collection. To control for differences in urine output and dilution, urine creatinine is typically measured. Although a 24-hour urine collection is considered an optimal sample because of fluctuations in excretion rates, most exposure studies have used a first morning void or a random sample because of the ease of collection. Under steady state exposure conditions⁴ random or spot urine results have correlated well with 24-hour results.

Testing for speciated urinary arsenic is preferable to testing for total urinary arsenic because the speciated forms can distinguish between exposure to inorganic arsenic and its metabolites and exposure to the relatively non-toxic forms of organic arsenic commonly found in seafood

⁴ Steady state conditions exist when the chemical level in an environmental medium exhibits only negligible change over a long period.

[Kallman 1990]. Because urine arsenic measurements reflect only recent exposure (within a few days), this measurement provides only a small window for assessment of arsenic exposure.

Inorganic urinary arsenic levels in unexposed individuals are normally less than 10 micrograms per liter ($\mu\text{g/L}$). Total arsenic levels below 50 $\mu\text{g/L}$ are expected for individuals without occupational or dietary exposures [NRC 1999]. Seventeen Superior residents provided a urine sample to test for urinary arsenic [MCEHP 2002d]. All 17 residents had urine arsenic levels below the detection limit of 5 $\mu\text{g/L}$.

Based on the available sampling data, results show that Superior residents were not exposed to unusual arsenic concentrations a few days (2–3 days) prior to their urine collection. However, these urinary results do not represent peak exposure levels to soil, sediment and waste tailings, such as those that might occur in the summer when outdoor activities occur. Therefore, despite the reported urine levels, arsenic exposures cannot be addressed on the basis of the results of the urine samples collected during February 2002 because of the timing of the collection.

4.4.2 Blood Lead

To measure lead exposure, the whole blood lead concentration is the single most useful laboratory test as it is indicative of recent exposure. However, it does not necessarily reflect the magnitude of external exposures such as ingestion of lead in soil. This is because blood lead levels are the product of both internal and external exposures.

Internal exposure results from the constant re-equilibration of lead between the bone (the primary storage compartment for lead) and the blood pool (primarily the erythrocytes or red blood cells). In persons with a high body burden of lead, redistribution of lead from bone into the bloodstream can keep blood lead levels elevated for years after external exposure stops. In such cases, the correct interpretation of a single blood lead test could require independent sources of information on the currency, frequency, and intensity of external exposure [Kosnet 2001].

The Centers for Disease Control and Prevention (CDC) considers a blood lead level (BLL) of 10 micrograms per deciliter ($\mu\text{g/dL}$) to be a level of concern for children. In February 2002, there were 66 Superior residents who were tested for lead exposure. All blood lead concentrations were below 10 $\mu\text{g/dL}$ [MCEHP 2002d]. Table 10, Appendix B, provides the BLLs for the various age groups.

However, as stated for the urine arsenic results, these blood lead results do not represent peak exposure levels to soil, sediment and waste tailings, such as those that might occur in the summer when outdoor activities occur. Therefore, despite the reported BLLs, external lead exposures cannot be addressed on the basis of the results of the blood samples collected during February 2002 because of the timing of the collection.

5 Public Health Evaluation

In this section, ATSDR addresses the question of whether exposure to contaminants at the concentrations detected would result in adverse health effects. While the relative toxicity of a chemical is important, the human body's response to a chemical exposure is determined by several additional factors. These factors include

- the concentration (how much) of the chemical the person was exposed to,
- the amount of time the person was exposed (how long), and

- the way the person was exposed (through breathing, eating, drinking, or direct contact with something containing the chemical).

Lifestyle factors (for example, occupation and personal habits) have a major impact on the likelihood, magnitude, and duration of exposure. Individual characteristics such as age, sex, nutritional status, overall health, and genetic constitution affect how a human body absorbs, distributes, metabolizes, and eliminates a contaminant. A unique combination of all these factors will determine the individual's physiologic response to a chemical contaminant and any harmful health effects the individual may suffer as a result of the chemical exposure.

As part of its evaluation, ATSDR derived exposure doses for children and adults. Estimating an exposure dose requires identifying how much, how often, and how long a person may come in contact with some concentration of the contaminant in a specific medium (like water and soil). Exposure doses help determine the extent to which an exposure might be associated with harmful health effects.

For this public health assessment, ATSDR made several assumptions with regard to site-specific exposure scenarios.

- For soil exposures, ATSDR assumed that residents have direct contact with and incidental ingestion of contaminated soil, sediment and waste tailings on the mine site, on the Flat Creek floodplain, and within the town of Superior.
- Because ATSDR expects that the mill site and floodplain area are only used sporadically for recreational activities, the agency assumed a short-term recreational exposure scenario would apply. And, although it is more likely that visitors to the area (hunters, hikers, etc.) moved around, for the short-term scenario, ATSDR assumed these visitors remain in the same location for several consecutive days.
- For the town of Superior, ATSDR evaluated two soil exposure scenarios—a pre-removal scenario and a post-removal scenario. For both scenarios, ATSDR evaluated short-term exposures and chronic exposures. And, although the ground may be frozen and/or covered in snow during the colder months, for the chronic exposure evaluation, ATSDR assumed residents were exposed to contaminated soil every day throughout the entire year.
- For surface water exposures, ATSDR assumed dermal contact and incidental ingestion occurs during wading and fishing in Flat Creek. For the family with a Flat Creek intake, ATSDR assumed daily ingestion exposure.
- For groundwater exposures, ATSDR assumed daily ingestion exposure.

The specific equations and default assumptions used to estimate exposure doses are provided in Appendix C. Of note, ATSDR typically used *maximum* chemical concentrations to calculate doses for acute exposures and *average* chemical concentrations to calculate doses for chronic exposures.

Two key steps in ATSDR's analysis involve (1) comparing the estimated site-specific exposure doses with observed effect levels reported in critical studies and (2) carefully considering study parameters in the context of site exposures [ATSDR 2005b]. This analysis requires the examination and interpretation of reliable substance-specific health effects data. This includes

reviews of epidemiologic (human) and experimental (animal) studies. These studies are characterized within ATSDR's toxicological chemical-specific profiles. Each peer-reviewed chemical profile identifies and reviews the key literature that describes a hazardous substance's toxicologic properties. ATSDR also reviews more recently released studies discussed in the scientific literature that may not have been captured in our toxicological profiles to ensure that our public health evaluations are based on the most current scientific knowledge.

Of note, substance-specific health effects data are generally expressed in terms of "ingested dose" rather than "absorbed dose." With regard to heavy metal exposure in soil, however, the distinction between ingested dose and absorbed dose is important. In general, ingestion of a metal in contaminated soil may be absorbed into the body to a much lesser extent than when the metal is ingested in drinking water or food.

Overall, assessing the relevance of available epidemiologic and experimental studies with respect to site-specific exposures requires both technical expertise and professional judgment. Because of uncertainties regarding exposure conditions and the harmful effects associated with environmental levels of exposure, definitive answers about whether health effects actually will or will not occur are not always possible. However, providing a framework that puts site-specific exposures and the potential for harm in perspective is possible and is one of the primary goals of ATSDR's public health evaluation process [ATSDR 2005b].

For the Flat Creek IMM site, the chemicals antimony, arsenic, cadmium, iron, lead, manganese, and thallium were detected above relevant health-based CVs in soil; the chemicals antimony, arsenic, cadmium, lead, manganese, and zinc were detected above relevant health-based CVs in water. In the following text, ATSDR provides a summary of the relevant epidemiologic and experimental information for antimony, arsenic, cadmium, iron, lead, manganese, thallium, and zinc. ATSDR then provides its public health evaluation of each chemical.

5.1 Antimony

Antimony is a silvery white metal found naturally in the earth's crust. It can enter the environment during mining processes, and small amounts can be released from incinerators and coal-burning power plants. Because items made from pure antimony are easily broken, it is usually mixed with other metals to form alloys, which are used in lead storage batteries, solder, sheet and pipe metal, bearings, castings, type metal, ammunition, and pewter [ATSDR 1992a]. Although antimony can exist in several forms, most analytical methods used by scientists to test for the presence of antimony in the environment do not distinguish the form of antimony [ATSDR 1992a].

Once in the environment, antimony cannot degrade, but it can change forms or become attached to or separated from particles. Soil usually contains less than 1 ppm of antimony [ATSDR 1992a]. The U.S. Geological Survey (USGS) reports an average of 0.66 ppm antimony in soil and a range of less than 1 to 8.8 ppm in uncontaminated areas within the contiguous United States [Shacklette and Boerngen 1984]. Much higher concentrations have been found in soil at hazardous waste sites that process antimony [View 1989]. Maximum concentrations of antimony were detected in waste tailings at concentrations of up to 4,500 ppm on the Iron Mountain Mine and Mill site and 2,360 ppm at a Superior residence [EPA 2002a].

As a natural constituent of soil, antimony is transported into streams and waterways from natural weathering of soil and from human sources. However, antimony has a low occurrence in ambient

water. It does not evaporate and only a small amount will dissolve in water. In a water monitoring survey by the Water Resources Division of the USGS, antimony was detected in less than 7% of the samples [Eckel and Jacob 1988]. The concentration of antimony in surface water is very low, usually less than 5 ppb [ATSDR 1992a]. However, antimony was detected in surface water at maximum concentrations of 472 ppb in Hall Gulch by the mine adit and 42 ppb in Flat Creek.

Because antimony is found naturally in the environment, the general population is exposed to low levels of it every day, primarily in food, drinking water, and air [ATSDR 1992a]. Studies on oral, dermal, and inhalation exposures to antimony in humans are limited. Evidence from studies on animals suggests that gastrointestinal absorption of antimony is 2–7% [Anonymous 1994, Dieter 1992, Felicetti et al. 1974, Gerber et al. 1982]. Gastrointestinal absorption of antimony is not expected to be above 10%, even though quantitative information on the absorption of antimony is not available for humans [ATSDR 1992a]. A few hours after exposure, a small amount of antimony enters the bloodstream and mostly distributes to the liver, lungs, intestines, and spleen. Antimony then leaves the body in urine and feces over several weeks. Consuming large quantities (19 ppm) may induce vomiting, which prevents most of the antimony from entering the bloodstream [ATSDR 1992a]. The bioavailability of antimony depends on the form of antimony present. For example, antimony incorporated in mineral lattices is inert and unlikely to be bioavailable [ATSDR 1992a, Dieter 1992, Flynn et al. 2003].

EPA's oral reference dose (RfD) for antimony of 0.0004 milligrams per kilogram per day (mg/kg/day) is based on a study in which health effects (decreased life span) were seen in rats exposed to 0.35 mg/kg/day of antimony in their drinking water [Schroeder et al. 1970]. EPA's confidence in the RfD is low because only one species was used, only one dose level was used, no no-observed-effect level (NOEL) was determined, and gross pathology and histopathology were not well described [EPA 2008]. ATSDR did not derive a chronic minimal risk level (MRL) from this study because ATSDR does not consider decreased life span in rats an appropriate basis for an MRL [ATSDR 1992a]. Of note, chronic MRLs, like RfDs, are an estimate of the daily human exposure to a substance that is likely to be without appreciable risk of noncarcinogenic adverse health effects.

The lowest intermediate LOAEL reported in ATSDR's *Toxicological Profile for Antimony* [ATSDR 1992a] is 0.0748 mg/kg/day, which is the dose that caused an adverse cardiovascular effect (decreased hypotensive response) in newborn rats and decreased maternal weight gain [Angrisani 1988, Marmo et al. 1987, Rossi et al. 1987]. More recent information found a NOAEL of 500 ppb antimony in drinking water, equivalent to an average intake of 0.06 mg/kg/day, which was established on the basis of the histological and biochemical changes in a 90-day exposure animal study [Poon 1998]. A human exposure study reported for acute exposures in the toxicologic literature was the result of people becoming acutely ill (stomach pains, colic, nausea, and vomiting) shortly after drinking lemonade contaminated with antimony. Their dose was approximately 0.5 mg/kg/day [Dunn 1928, Monier-Williams 1934].

The Department of Health and Human Services (DHHS) and the EPA have not classified antimony as to its human carcinogenicity [ATSDR 1992a].

For this public health assessment, ATSDR derived exposure doses for Superior residents exposed to antimony in the soil and water based on its pathway evaluation. In the following text, ATSDR provides a brief description of the exposure scenario and then compares the estimated,

site-specific antimony exposure doses with the observed effect levels reported in the critical studies.

5.1.1 Soil

As stated previously, residents are exposed to antimony in sediment and waste tailings when they engage in hunting, fishing, wading, hiking, and other recreational activities on the Iron Mountain Mine and Mill site and along the Flat Creek floodplain area. For this short-term exposure scenario, children's exposure doses based on the maximum antimony level in sediment and waste tailings are 0.02 mg/kg/day and 0.06 mg/kg/day, respectively. These doses are an order of magnitude (or 10 times) below the lowest LOAEL (0.5 mg/kg/day) reported for acute exposures (up to 14 days). Adult exposures to the maximum antimony levels in sediment and waste tailings, as well as child and adult exposures to average antimony levels, are one to two orders of magnitude (or 10 to 100 times) below antimony's lowest LOAEL for acute exposures. Therefore, exposure to antimony found in the sediment and waste tailings in these areas would not be expected to be of health concern to children or adults (1) because antimony is not expected to be 100% bioavailable as assumed in ATSDR's dose calculations, and (2) because antimony concentrations are below known effect levels for acute exposure. Also, because the contaminated areas at the mine site and floodplain area are posted with warning signs, the frequency of recreational activities occurring in these areas is expected to be low.

In addition, residents are exposed to antimony in waste tailings used as fill in the town of Superior. Prior to removal actions, children's exposure doses based on the maximum levels of antimony in soil were 0.01 mg/kg/day at the fairgrounds and 0.03 mg/kg/day at a residence. These doses are an order of magnitude below antimony's acute LOAEL (0.5 mg/kg/day), but just slightly below the lowest LOAEL for intermediate exposure (0.0748 mg/kg/day). However, ATSDR's estimated doses do not consider that gastrointestinal absorption of antimony is not expected to be above 10% and that antimony incorporated in mineral lattices is inert. When ATSDR used an antimony bioavailability factor of 10% to estimate soil antimony exposure doses, all doses were an order of magnitude or more below these LOAELs. Following removal actions, a child's exposure dose (0.0008 mg/kg/day) based on the maximum level of antimony in Superior soil is also several orders of magnitude below these LOAELs. The exposure doses for adults are lower still. As such, harmful health effects are not expected to result from past, current, and future exposures to antimony in town soil.

5.1.2 Surface Water

A past, current, and future completed exposure pathway to surface water exists for recreational users of Flat Creek. Children's exposure doses based on the maximum antimony level (42 ppb) in Flat Creek surface water are not at a level of public health concern because the estimated child dose (0.0004 mg/kg/day) is several orders of magnitude below antimony's acute and intermediate LOAELs. Likewise, harmful health effects are not expected for adults following exposure to antimony in Flat Creek surface water while wading and fishing downstream of the mine site because the estimated exposure doses are several orders of magnitude below these LOAELs.

In addition to recreational use, one family obtains part of their household water supply from a Flat Creek intake [EPA 2002a]. For this family, a past, current, and future completed exposure pathway exists through activities such as drinking, showering, bathing, cooking, and washing dishes with the surface water. The one surface water sample collected from their Flat Creek

intake contained antimony at 12 ppb, which is above EPA's MCL (6 ppb). EPA's MCLs are the maximum allowable concentration for specific chemicals in public drinking water. MCLs are considered protective of public health over a lifetime of exposure. Although this antimony level exceeds EPA's MCL, harmful health effects are not expected because the estimated child dose (0.001 mg/kg/day) and estimated adult dose (0.0003 mg/kg/day) are one to two orders of magnitude below antimony's LOAELs for acute and intermediate exposures. The maximum antimony level (42 ppb) detected in Flat Creek downstream of the mine site also results in estimated child and adult doses that are an order of magnitude below these LOAELs. Even though only one sample was available for the family's Flat Creek intake, based on that one sample as well as the other creek water samples, exposure to antimony levels in the creek would not be expected to result in harmful health effects. Although ATSDR determined that harmful health effects are unlikely to occur, efforts to reduce drinking water exposures are considered prudent public health practice when chemical levels are above EPA's MCL.

Shallow, reddish-brown surface water exists in the Hall Gulch mine adit area. ATSDR would not expect people to occasionally drink, or wade in, this surface water. Without exposure, adverse health effects are not expected. However, because antimony exposures could potentially be of public health concern if the water were to be ingested, ATSDR recommends Hall Gulch continue to be posted with a warning sign informing the public that water coming out of the adit has levels of metals that may pose a health risk.

5.1.3 Groundwater

Past, current and future use of the three town of Superior wells is a completed exposure pathway. However, antimony was detected in only 10 of 43 distribution samples, and exceeded ATSDR's child RMEG and EPA's MCL in just one of those samples. Harmful health effects are not expected because the estimated child dose (0.003 mg/kg/day) and estimated adult dose (0.0009 mg/kg/day), which assume daily, continuous exposure to the maximum detected antimony concentration, are one to two orders of magnitude below antimony's acute and intermediate LOAELs.

As stated previously, Flat Creek Spring arises from area groundwater and the town of Superior sampled the spring water between 1993 and 1998, during which time it served as the primary source for the PWS. Spring water samples ranged from not detected to 34 ppb, with an average of 28 ppb. Levels were consistently above EPA's MCL, a regulatory guideline. Therefore, use of the spring as a drinking water source was discontinued. With regard to past drinking water exposures, ATSDR determined harmful health effects are not expected because the estimated child dose (0.003 mg/kg/day) and estimated adult dose (0.0008 mg/kg/day) are one to two orders of magnitude below antimony's acute and intermediate LOAELs.

Although ATSDR determined that harmful health effects are unlikely to occur, eliminating drinking water exposure to the spring is considered prudent public health practice because antimony levels were consistently above a regulatory guideline. Future use of the Flat Creek Spring represents a potential exposure pathway. ATSDR recommends testing of the spring to ensure antimony levels meet regulatory guidelines before using it as an emergency supply.

Private wells also represent a past, current and future completed exposure pathway. Only one private well sample was collected and it contained antimony at 24 ppb. Although this antimony level exceeds a regulatory guideline (EPA's MCL), harmful health effects are not expected because the estimated child dose (0.002 mg/kg/day) and estimated adult dose (0.0007

mg/kg/day) are one to two orders of magnitude below antimony's acute and intermediate LOAELs. As stated previously though, ATSDR considers efforts to reduce drinking water exposures prudent public health practice when chemical levels are above an EPA MCL.

5.2 Arsenic

Arsenic, a naturally occurring element, is widely distributed in the Earth's crust, which contains about 3.4 ppm arsenic [Wedepohl 1991]. Most arsenic compounds have no smell or distinctive taste. Although elemental arsenic sometimes occurs naturally, arsenic is usually found in the environment in two forms—inorganic (arsenic combined with oxygen, chlorine, and sulfur) and organic (arsenic combined with carbon and hydrogen). Sometimes, the specific form of arsenic present in the environment is not determined. Therefore, what form of arsenic a person may be exposed to is not always known.

Most simple organic forms of arsenic are less harmful than the inorganic forms [ATSDR 2007a]. Once in the environment, arsenic cannot be destroyed; it can only change forms or become attached to or separated from particles (e.g., by reacting with oxygen or by the action of bacteria in soil). Some forms of arsenic may be so tightly attached to particles or embedded in minerals that they are not taken up by plants and animals.

Arsenic is released to the environment through natural sources such as wind-blown soil and volcanic eruptions. However, anthropogenic (man-made) sources of arsenic release much higher amounts of arsenic than natural sources. These anthropogenic sources include nonferrous metal mining and smelting, pesticide application, coal combustion, wood combustion, and waste incineration. About 90% of all commercially produced arsenic is used to pressure-treat wood [ATSDR 2007a]. In the past, arsenic was widely used as a pesticide; in fact, some organic arsenic compounds are still used in pesticides.

Drinking water is an important factor of arsenic exposure. The concentrations of arsenic in water are usually less than 10 ppb. The average value was 2.4 ppb in a study of tap water from 3,834 U.S. residences [EPA 1982b]. A survey of 293 stations on major rivers in the United States found median arsenic levels to be 1 ppb [Smith et al. 1987]. However, the concentrations can vary considerably depending on local natural or anthropogenic sources. In 2004, arsenic in surface water samples recorded in the EPA's STORET (short for STORage and RETrieval) database ranged from not detected to 1,700 ppb [EPA 2005]. Groundwater is far more likely to contain high levels of arsenic than surface water. Arsenic levels in groundwater average about 1–2 ppb, except where volcanic rock and sulfidic mineral deposits are high in arsenic (e.g., in some western states arsenic levels up to 3,400 ppb have been observed) [IARC 1980, Page 1981, Robertson 1989, Welch et al. 1988]. In western mining areas, concentrations up to 48,000 ppb have been reported in groundwater [Welch et al. 1988]. Arsenic levels in Flat Creek surface water ranged from 3.8–6 ppb. For the town of Superior, arsenic levels in three distribution system samples ranged from not detected to 1 ppb.

People may also be exposed through incidentally ingesting soil containing arsenic. Arsenic concentrations for uncontaminated soils generally range from 1–40 ppm, with a mean of 5 ppm [ATSDR 2007a]. Arsenic concentrations in soils from various countries range from 0.1 to 50 ppm and can vary widely among geographic regions. USGS reports a mean of 7.2 ppm and a range of less than 0.1–97 ppm in the United States [Shacklette and Boerngen 1984]. For Montana, MDEQ reports a mean of 29 ppm and a range of 0.94–187 ppm [MDEQ 2005]. Higher arsenic levels may be found in the vicinity of arsenic-rich geological deposits, some mining and

smelting sites, or agricultural areas where arsenic pesticides had been applied in the past. For example, arsenic concentrations up to 27,000 ppm were reported in soils contaminated with mine or smelter wastes [EPA 1982a]. The maximum arsenic concentration in Flat Creek sediments affected by waste tailings was 40,200 ppm. Waste tailings on the mine site had a maximum arsenic concentration of 9,350 ppm and contaminated soil from a residential yard contained arsenic at 2,620 ppm.

Ingestion of arsenic-contaminated water and soil are ways that arsenic can enter the body. Dermal exposure to arsenic is usually not of concern because only a small amount will pass through skin and into the body (4.5% of inorganic arsenic in soil) [Wester et al. 1993]. The metabolism of inorganic arsenic has been extensively studied in humans and animals. Several studies in humans indicate that arsenic is well absorbed across the gastrointestinal tract (approximately 95% absorption for inorganic arsenic compounds and 75–85% for organic arsenic compounds) [Bettley and O'Shea 1975, Buchet et al. 1981, Marafante et al. 1987, Zheng et al. 2002]. Once in the body, the liver changes (i.e., through methylation) some of the inorganic arsenic to less harmful organic forms that are more readily excreted in urine. Most forms of organic arsenic appear to undergo little metabolism. Both inorganic and organic forms of arsenic leave the body in urine. It is estimated that more than 75% of the absorbed arsenic dose is excreted in urine [Marcus and Rispin 1988]. Studies have shown that 45–85% of arsenic is eliminated within one to three days [Apostoli et al. 1999, Buchet et al. 1981, Crecelius 1977, Tam et al. 1979]. However, there appears to be an upper-dose limit to this mechanism working successfully to reduce arsenic toxicity [ATSDR 2007a].

As noted above, water-soluble forms of inorganic arsenic are well absorbed. Ingesting less soluble forms of arsenic results in reduced absorption. Studies in laboratory animals show that arsenic in soil is only one-half to one-tenth as bioavailable as soluble arsenic forms [Casteel et al. 1997, Freeman et al. 1993, Freeman et al. 1995, Groen et al. 1994, Rodriguez et al. 1999]. In one study, approximately 80% of the arsenic from ingested soil was eliminated in the feces compared with 50% of the soluble oral dose [Freeman et al. 1993]. The bioavailability of arsenic in soil may be reduced due to low solubility and inaccessibility [Davis et al. 1992]. Most of the bioavailable arsenic in water and soil is expected to be present as inorganic arsenic (trivalent arsenic and pentavalent arsenic, specifically) [Health Canada 1993].

ATSDR's acute oral MRL⁵ (0.005 mg/kg/day) is based on a study in which 220 people in Japan were exposed to arsenic contaminated soy sauce for a 2–3 week period. The dose was estimated to be 0.05 mg/kg/day. This is considered the LOAEL. Facial edema and gastrointestinal symptoms (nausea, vomiting, and diarrhea) were considered to be the critical effects seen at this dose [Mizuta et al. 1956]. The MRL is further supported by the case of a man and woman in upstate New York who experienced gastrointestinal symptoms after drinking arsenic-tainted water at an estimated dose of 0.05 mg/kg/day [Franzblau and Lilis 1989].

The chronic oral MRL (0.0003 mg/kg/day) is based on a study in which a large number of farmers (both male and female) were exposed to high levels of arsenic in well water in Taiwan. EPA's oral RfD is also 0.0003 mg/kg/day [EPA 2008]. A clear dose-response relationship was observed for characteristic skin lesions. A control group consisting of 17,000 people was exposed to 0.0008 mg/kg/day and did not experience adverse health effects. This is considered to be the NOAEL. Hyperpigmentation and keratosis of the skin were reported in farmers exposed to

⁵ The acute oral MRL is considered provisional because it is based on a serious LOAEL.

0.014 mg/kg/day (less serious LOAEL). Those exposed to 0.038–0.065 mg/kg/day experienced an increased incidence of dermal lesions [Tseng et al. 1968, Tseng 1977]. The MRL is supported by a number of well conducted epidemiological studies that identify reliable NOAELs and LOAELs for dermal effects [Borgoño and Greiber 1972, Cebrian et al. 1983, EPA 1981, Guha Mazumder et al. 1988, Haque et al. 2003, Harrington et al. 1978, Valentine et al. 1985, Zaldívar 1974]. Collectively, these studies indicate that the threshold dose for dermal effects (ex., hyperpigmentation and hyperkeratosis) is approximately 0.002 mg/kg/day.

DHHS, the International Agency for Research on Cancer (IARC), and EPA have all independently determined that inorganic arsenic is carcinogenic to humans. There is convincing evidence from a large number of epidemiological studies and case reports that ingestion of inorganic arsenic increases the risk of developing skin cancer [Alain et al. 1993, Beane Freeman et al. 2004, Bickley and Papa 1989, Cebrián et al. 1983, Chen et al. 2003, Hauptert et al. 1996, Hsueh et al. 1995, Lewis et al. 1999, Lühtrath 1983, Mitra et al. 2004, Morris et al. 1974, Sommers and McManus 1953, Tay and Seah 1975, Tsai et al. 1998, Tsai et al. 1999, Tseng 1977, Tseng et al. 1968, Zaldívar 1974, Zaldívar et al. 1981]. A report by the National Research Council suggests that the risks calculated based on increases in incidence of lung and bladder cancers may be greater than those calculated based on incidences of skin cancer [NRC 2001].

For this health consultation, ATSDR derived exposure doses for Superior residents exposed to arsenic in the soil and water based on the pathway evaluation. Of note, arsenic's site-specific bioavailability factors were not known. As a conservative measure, ATSDR used an arsenic bioavailability factor of 50% for soil exposures and 100% for water exposures when estimating site-specific arsenic exposure doses. In the following text, ATSDR provides a brief description of the exposure scenarios and then compares the estimated, site-specific arsenic exposure doses with the observed effect levels reported in the critical studies.

5.2.1 Soil

As stated previously, a past, current, and future completed exposure pathway to surface soil, sediment, and waste tailings exists for people engaged in hunting, fishing, wading, hiking, and other recreational activities on the Iron Mountain Mine and Mill site and along the Flat Creek floodplain area. In addition, a past, current, and future completed exposure pathway exists to waste tailings used as fill in the town of Superior.

5.2.1.1 Iron Mountain Mine and Mill Site and Flat Creek Floodplain

For the short-term exposure scenario, a child's exposure dose based on the maximum arsenic level in sediment is at a level of public health concern because the estimated child dose (0.3 mg/kg/day) exceeds arsenic's acute LOAEL (0.05 mg/kg/day). In addition, children's exposure doses based on the maximum arsenic level in waste tailings, the average arsenic level in sediment, and the average arsenic level in waste tailings are at a level of potential public health concern for short-term exposure because the estimated doses (0.06 mg/kg/day, 0.02 mg/kg/day, and 0.02 mg/kg/day, respectively) are comparable to the acute LOAEL. The estimated adult dose (0.03 mg/kg/day) based on the maximum arsenic level in sediment also is comparable to this LOAEL.

Because the arsenic contaminated areas at the mine site and floodplain area are posted with warning signs, ATSDR would expect the frequency of recreational activities occurring in these areas to be low. However, if children and adults were to repeatedly visit these areas of elevated

arsenic concentrations and participate in contact-intensive activities, it is plausible that they may experience transient harmful effects (nausea, vomiting, and diarrhea) following their exposures. ATSDR recommends residents take prudent public health measures to reduce their ingestion or inhalation of dust and soil.

With regard to carcinogenicity, the frequency of visitors to the mine site and floodplain area is expected to be low. Therefore, visitors are not chronically exposed to arsenic-contaminated sediment and waste tailings. Occasional exposure to arsenic in the sediment and waste tailings on the mine site and floodplain area would not be expected to result in cancer.

5.2.1.2 *Town of Superior*

Prior to removal actions, children's exposure doses based on the maximum levels of arsenic in soil at various town locations such as at a residence (2,620 ppm arsenic) and at the high school track (2,110 ppm arsenic), were at a level of public health concern for short-term exposures because the estimated doses were comparable to arsenic's acute LOAEL. Prior to removal actions, if children lived or visited these areas of elevated arsenic soil concentrations and participated in contact-intensive activities, it is plausible that they may have experienced transient harmful effects (nausea, vomiting, and diarrhea) following their short-term exposures.

Prior to removal actions, children's chronic exposure doses based on average soil arsenic levels in some areas of town, such as at the high school track (904 ppm arsenic), exceeded the threshold dose for dermal effects of approximately 0.002 mg/kg/day. Therefore, ATSDR considers these past arsenic levels to have been of public health concern. Because the 2002 TCRA removed contaminated soil from these areas, current and future exposures to arsenic are not expected to be at levels of health concern in these areas. However, some uncertainty exists about the levels of arsenic that remain. Additional characterization (e.g., post-confirmation sampling and analysis) of these areas would be needed to ensure that removal efforts were successful.

To provide some perspective on the potential that soil contamination in Superior may currently be of health concern in some areas, ATSDR *estimated* potential current contamination levels using data from the 2002 sampling event. Based on these estimates, acute and chronic exposures to arsenic for most residential and non-residential areas are not at levels of health concern because the exposure doses are an order of magnitude or more below known adverse effect levels. However, the potential exists for additional properties in town that were not sampled to contain waste tailings material with elevated levels of arsenic.

In addition, children's chronic exposure doses based on the maximum arsenic levels at two residences (175 ppm and 392 ppm) currently are at and approaching the threshold dose (0.002 mg/kg/day) for dermal effects. ATSDR considers chronic exposure to these arsenic levels in residential yards of public health concern. Of note, it is not clear whether these maximum arsenic levels are representative of average arsenic levels that children might be exposed when playing in these yards. Additional characterization of these yards is needed to determine whether the maximum arsenic concentrations are truly representative. As a protective measure, ATSDR suggests that if children live at or visit these two residences, parents should monitor their children's behavior while playing outdoors to prevent their children from intentionally or inadvertently eating soil. ATSDR is working closely with EPA and local agencies to ensure these residents are made aware of ATSDR's recommendations.

With regard to carcinogenic risk, most properties are currently below a level of potential concern for cancer. Although the two properties mentioned previously contain elevated arsenic levels (175 ppm and 392 ppm), the harmful health effects observed in the studies on arsenic ingestion involved daily, long-term ingestion of elevated arsenic levels in drinking water. Ingestion of large amounts of soil would most likely not occur 365 days a year for life. ATSDR does not consider ingestion of arsenic in soil to be of health concern for cancer.

Overall, prior to removal actions, levels of arsenic in soil for some town properties were of public health concern for children and adults. Currently, two residential yards contain arsenic at levels of public health concern for a chronic exposure scenario. Although not all areas of town have been characterized, the arsenic levels detected at most residential and non-residential areas are currently not at levels of health concern for children or adults because the estimated exposure doses (both acute and chronic) are an order of magnitude or more below known adverse effect levels.

Because data are limited and the waste tailings contain elevated levels of arsenic, any residents who suspect waste tailings to be present in their yard should monitor their children outdoors to prevent their children from intentionally or inadvertently eating soil. Additional activities residents may do to reduce exposure to contaminated soil are discussed in Appendix D.

5.2.2 Surface Water

A past, current, and future completed exposure pathway to surface water exists for recreational users of Flat Creek. Children's exposure based on the maximum arsenic level in Flat Creek is not at a level of public health concern because the estimated child dose (0.00006 mg/kg/day) is three orders of magnitude below arsenic's acute LOAEL (0.05 mg/kg/day) and two order of magnitude below the threshold dose (0.002 mg/kg/day) for dermal effects. The exposure doses for adults are lower still. Therefore, harmful health effects are not expected for children and adults following exposure to arsenic in Flat Creek surface water while wading and fishing downstream of the mine site because the estimated exposure doses are several orders of magnitude below the lowest LOAELs.

In addition to recreational use, one family obtains part of their household water supply from a Flat Creek intake [EPA 2002a]. For this family, a past, current, and future completed exposure pathway exists through activities such as drinking, showering, bathing, cooking, and washing dishes with the surface water. The one surface water sample collected from their Flat Creek intake did not show a detectable level of arsenic. Based on this sample and the additional 24 surface water samples collected from various points in Flat Creek downstream from the mine site, it appears arsenic levels in the creek are below EPA's MCL. Of note, children's exposure based on the maximum arsenic level in Flat Creek is not at a level of public health concern because the estimated child dose (0.0006 mg/kg/day) is two orders of magnitude below arsenic's acute LOAEL (0.05 mg/kg/day) and an order of magnitude below the threshold dose (0.002 mg/kg/day) for dermal effects. ATSDR does not consider the arsenic levels found in creek water to be of health concern for cancer. Although only one sample was available for the family's Flat Creek intake, based on that sample as well as the other creek water samples, exposure to the arsenic levels in the creek would not be expected to result in harmful health effects.

Shallow, reddish-brown surface water exists in Hall Gulch. ATSDR would not expect people to occasionally drink, or wade in, this surface water. Without exposure, adverse health effects are not expected. However, because arsenic exposure doses would be at levels of public health

concern if the water were to be frequently ingested, ATSDR recommends Hall Gulch continue to be posted with a warning sign informing the public that water coming out of the adit has levels of metals that may pose a health risk.

5.2.3 Groundwater

Past, current and future use of the three town of Superior wells is a completed exposure pathway. Private wells also represent a past, current and future completed exposure pathway. Future use of the Flat Creek Spring represents a potential exposure pathway. Exposure activities include drinking, showering, bathing, cooking, and washing dishes with the groundwater.

Children's exposure based on the maximum arsenic level in groundwater is not at a level of public health concern because the estimated child dose (0.0004 mg/kg/day) is two orders of magnitude below arsenic's acute LOAEL (0.05 mg/kg/day) and one order of magnitude below the threshold dose (0.002 mg/kg/day) for dermal effects. The exposure doses for adults are lower still. ATSDR does not consider the arsenic levels found in groundwater to be of health concern for cancer. Furthermore, although the data for arsenic in groundwater are limited, particularly for private wells (as only one sample exists), no concentrations have exceeded EPA's MCL. As such, exposure to arsenic in groundwater would not be expected to result in harmful health effects.

5.3 Cadmium

Cadmium is an element that occurs naturally in the earth's crust. It is not usually present in the environment as a pure metal, but as a mineral combined with other elements such as oxygen (cadmium oxide), chlorine (cadmium chloride), or sulfur (cadmium sulfate, cadmium sulfide) [ATSDR 2008b]. Cadmium compounds are typically solids that dissolve in water to varying degrees (e.g., the chlorides and sulfates easily dissolve in water). Cadmium metal changes forms in the environment, but it does not disappear. It has no recognizable odor or taste.

Natural processes, such as weathering of minerals, forest fires, and volcanic emissions, release small amounts of cadmium to the environment. However, anthropogenic sources (e.g., mining and smelting operations, fuel combustion, disposal of metal-containing products, and application of phosphate fertilizer or sewage sludges) release ten times as much cadmium [Elinder 1985, Elinder 1992, IARC 1993]. Cadmium is widely used in industrial and consumer products, including batteries, pigments, metal coatings, plastics, and some metal alloys.

Cadmium concentrations in unpolluted soil range from 0.01–1 ppm in soils of nonvolcanic origin and up to 4.5 ppm in solids of volcanic origin [Eisler 1985]. The average level of cadmium in uncontaminated topsoil in the United States is about 0.25 ppm [EPA 1985a]. However, levels as high as 800 ppm have been found in soils from polluted areas [IARC 1993]. Maximum concentrations of cadmium were detected in waste tailings along Flat Creek at concentrations of 161 ppm and at a Superior residence at 46.3 ppm.

Concentrations of cadmium in natural surface water and groundwater are typically less than 1 ppb, but may be higher in waters from areas with cadmium-bearing mineral formations (e.g., concentrations could be >1,000 ppb in these locations) [Elinder 1985, Elinder 1992, Thornton 1992]. In 1987 and 1988, USGS reported cadmium concentrations in the Mississippi River ranging from 0.05 ppb to 8 ppb [Taylor et al. 1990]. In a survey of public drinking water supplies, the U.S. Public Health Service found that most of the supplies did not contain more than 1 ppb of cadmium. However, the average concentration was 3 ppb based on a handful of

supplies that contained greater than 10 ppb of cadmium [IARC 1993, Konz and Walker 1979]. For the Flat Creek IMM site, cadmium was detected in surface water at maximum concentrations of 21.8 ppb in Hall Gulch by the mine adit and 1.6 ppb in Flat Creek. In the distribution system, cadmium was found at a maximum concentration of 3 ppb.

Dermal exposure to cadmium is not known to affect human health because under normal conditions, virtually no cadmium can enter the body through the skin (less than 0.2% from soil) [Wester et al. 1992]. Generally, the main sources of cadmium exposure are through smoking cigarettes and, to a lesser extent, eating foods contaminated with cadmium. However, only about 5 to 10% of ingested cadmium is actually absorbed by the body; the majority is passed out of the body in feces [Kjellstrom et al. 1978, McLellan et al. 1978, Rahola et al. 1973]. Cadmium that is absorbed goes to the kidneys and liver. Because cadmium leaves the body slowly, once it is absorbed, it tends to remain in the body for years. The body changes most of the cadmium into a form that is not harmful, but if too much cadmium is absorbed, the liver and kidneys cannot convert all of it into the harmless form [Kotsonis and Klaassen 1978, Sendelbach and Klaassen 1988].

EPA derived separate RfDs for cadmium in drinking water (0.0005 mg/kg/day) and in food (0.001 mg/kg/day), based on data from many studies on the toxicity of cadmium in both humans and animals. The critical effect is significant proteinuria in humans chronically exposed to cadmium, using a NOAEL of 200 mg of cadmium per gram wet weight in the renal cortex and a toxicokinetic model assuming 2.5 or 5% cadmium absorption from food or water, respectively, and 0.01% cadmium excretion per day. This corresponds to estimated NOAELs of 0.01 mg/kg/day for food and 0.005 mg/kg/day for water [EPA 2008].

ATSDR conducted a meta-analysis of select environmental exposure dose-response studies examining the relationship between urinary cadmium and the prevalence of elevated levels of biomarkers of renal function in the derivation of its MRL [ATSDR 2008b]. ATSDR's chronic oral MRL (0.0001 mg/kg/day) is comparable to the EPA RfD for water (0.0005 mg/kg/day). In addition, ATSDR found that the animal studies identify several sensitive targets of toxicity following intermediate-duration exposure to cadmium, including skeletal mineralization in young female rats exposed for at least 3 months to 0.2 mg/kg/day of cadmium [Brzóška and Moniuszko-Jakoniuk 2005a, Brzóška et al. 2005b, Brzóška et al. 2005c].

Studies in humans and animals do not provide sufficient evidence to determine whether cadmium is a carcinogen via the oral route [ATSDR 2008b]. As a conservative approach, IARC has determined that cadmium is carcinogenic to humans. DHHS reasonably anticipates that cadmium is a carcinogen. EPA has determined that when inhaled, cadmium is a probable human carcinogen. Only one cancer effect level is reported in ATSDR's *Toxicological Profile for Cadmium*—increased rates of prostatic adenomas resulted in rats exposed to 3.5 mg/kg/day of cadmium in their food [ATSDR 2008b]. EPA does not have an oral cancer slope factor because there are no positive studies of orally ingested cadmium suitable for quantitation [EPA 2008].

For this health consultation, ATSDR derived exposure doses for Superior residents exposed to cadmium in the soil and water based on its pathway evaluation. In the following text, ATSDR provides a brief description of the exposure scenario and then compares the estimated, site-specific cadmium exposure doses with the observed effect levels reported in the critical studies.

5.3.1 *Soil*

Residents are exposed to cadmium in sediment and waste tailings when they engaged in hunting, fishing, wading, hiking, and other recreational activities on the mine site and along the floodplain area. For the short-term exposure scenario, children's exposure doses based on the maximum cadmium level in sediment and waste tailings are not at a level of public health concern because the estimated child doses (0.0007 mg/kg/day and 0.002 mg/kg/day, respectively) are two to three orders of magnitude below a known intermediate-duration effect level (0.2 mg/kg/day). Adult exposures are even lower. Therefore, for the short-term exposure scenario, harmful health effects are not expected to result from exposure to cadmium in sediment and waste tailings from the mill site and Flat Creek floodplain area.

In addition, residents are exposed to cadmium in waste tailings used as fill in the town of Superior. Prior to removal actions, children's exposure doses based on the maximum levels of cadmium in soil at the fairgrounds (0.0006 mg/kg/day) and a residence (0.0004 mg/kg/day) are several orders of magnitude below cadmium's known intermediate-duration effect level (0.2 mg/kg/day). These children's doses are also below EPA's estimated NOAELs of 0.01 mg/kg/day for food and 0.005 mg/kg/day for water. Following removal actions, children's exposure doses based on the maximum levels of cadmium in soil (0.0001 mg/kg/day) are also well below the known intermediate-duration effect levels and EPA's estimated NOAELs. The exposure doses for adults are lower still. In addition, ATSDR does not consider the cadmium levels found in soil to present an elevated carcinogenic risk. Overall, harmful health effects are not expected to result from past and current exposure to cadmium in town soil.

5.3.2 *Surface Water*

A past, current, and future completed exposure pathway to surface water exists for recreational users of Flat Creek and for the one family who obtains part of their household water supply from a Flat Creek intake. However, cadmium has not been detected in creek water above health-based comparison values. In addition, ATSDR does not consider the cadmium levels found in surface water to present an elevated carcinogenic risk because the cadmium levels are low and the estimated exposure doses are well below cancer effect levels. Therefore, harmful health effects are not expected for children and adults (1) following exposure to cadmium in Flat Creek surface water while wading and fishing downstream from the mine site and (2) following exposure from drinking, showering, bathing, cooking, and washing dishes with this surface water.

Cadmium was detected only once in surface water above a health-based comparison value in the Hall Gulch mine adit area. As stated previously, ATSDR consider Hall Gulch surface water an eliminated exposure pathway. Without exposure, adverse health effects are not expected.

5.3.3 *Groundwater*

Cadmium has not been detected above EPA's MCL in any samples from the three town of Superior wells, one private well, and Flat Creek Spring. Although one sample from the distribution system did exceed ATSDR's chronic child EMEG, chronic exposure to elevated cadmium levels is not occurring. Children's exposure dose (0.0003 mg/kg/day) based on this one sample's cadmium level is below cadmium's known intermediate-duration effect level (0.2 mg/kg/day) and is below EPA's estimated NOAELs of 0.01 mg/kg/day for food and 0.005 mg/kg/day for water. In addition, ATSDR does not consider the cadmium levels found in groundwater to present an elevated carcinogenic risk because the cadmium levels are low and the

estimated exposure doses are well below cancer effect levels. Therefore, harmful health effects are not expected.

5.4 Iron

Iron is a naturally occurring element in the environment. In fact, by weight it is the fourth most abundant element in the earth's crust [LANL 2003]. The most common iron ore is hematite, which frequently can be seen as black sand along beaches and stream banks. As a pure metal, iron is very reactive chemically and will rapidly corrode, especially in moist air or at high temperatures. It is hard and brittle, and is usually combined with other metals to form alloys, including steel.

Iron is an important mineral for human health, assisting in the maintenance of basic life functions. It combines with protein and copper to make hemoglobin, which transports oxygen in the blood from the lungs to other parts of the body. It also aids in the formation of myoglobin, which supplies oxygen to muscle tissues. Without sufficient iron, the body cannot produce enough hemoglobin or myoglobin to sustain life. Iron deficiency anemia is a condition that occurs when the body does not receive enough iron. The National Academy of Sciences' (NAS) recommended dietary allowance (RDA) for all age groups of men and postmenopausal women is 8 milligrams per day (mg/day); the RDA for premenopausal women is 18 mg/day [NAS 2001]. The U.S. Food and Drug Administration's (FDA) reference daily intake (RDI) for iron is 18 mg/day [Kurtzweil 1993]. NAS reports the median daily intake of dietary iron is roughly 11–13 mg/day for children 1–8 years old [NAS 2001].

From 1961 through the autumn of 1975, Shacklette and Boerngen (1984) collected background soil samples from 1,318 uncontaminated areas across the contiguous United States. The samples were taken at a depth of approximately 20 centimeters (7.9 inches) from locations about 80 kilometers (50 miles) apart. The average iron concentration was 26,000 ppm, and ranged from 100 to >100,000 ppm [Shacklette and Boerngen 1984]. Maximum concentrations of iron were detected in sediment affected by waste tailings near the Iron Mountain mine adit at 298,000 ppm and at a Superior residence at 63,200 ppm.

The body uses a homeostatic mechanism to keep iron burdens at a constant level despite variations in the diet [Eisenstein and Blemings 1998]. More iron is absorbed when the stores of iron in the body are low. When the body has adequate levels of iron, absorption decreases to help protect against iron toxicity [Bothwell et al. 1979, NAS 2001]. However, if chronically exposed to high levels of iron, the body does not have a mechanism to prevent excessive accumulation of iron [NAS 2001]. The type of iron consumed also influences absorption—absorption of heme iron⁶ ranges from 15–35%; whereas, absorption of nonheme iron⁷ ranges from 2–20% [Monson 1988, Tapiero et al. 2001]. Healthy adults typically absorb about 10–15% of dietary iron⁸, but this is influenced by several factors [Bothwell et al. 1979, Davidsson 2003, Hallberg et al. 1997, Miret et al. 2003, Monson 1988, NAS 2001, Sandberg 2002, Uzel and Conrad 1998]. Storage levels of iron influence iron absorption the most. No specific studies regarding dermal exposure to iron are available; but metals such as iron are typically not readily absorbed through the skin.

⁶ Heme iron is bound within the iron-carrying proteins (hemoglobin and myoglobin) found in meat, poultry, and fish.

⁷ Nonheme iron is less easily absorbed by the body and is influenced by the composition of the diet.

⁸ The percents presented for iron absorption refer to the amount of dietary iron that the body obtains and uses from food.

Acute iron poisoning has been reported in children less than 6 years of age who have accidentally overdosed on iron-containing supplements for adults. According to the FDA, doses greater than 200 mg per event could poison or kill a child [FDA 1997]. EPA's oral RfD for iron (0.7 mg/kg/day) is a provisional peer-reviewed value based on a study in 97 Swedish adult men and women who experienced gastrointestinal effects while taking iron supplements at doses of 1 mg/kg/day (i.e., 60 mg/day) [EPA 2006]. NAS found that several studies support a LOAEL between 50 and 120 mg/day for supplemental iron salts [Brock et al. 1985, Coplin et al. 1991, Frykman et al. 1994, Liguori 1993, Lokken and Birkeland 1979]. Based on these data, NAS calculated tolerable upper intake levels of 40 mg/day for children 7 months to 13 years old [NAS 2001]. Looker et al. [1988] evaluated dietary intake data collected as part of the Second National Health and Nutrition Examination Survey and found that no adverse health effects were associated with average iron intakes of 0.15–0.27 mg/kg/day. The relationship between iron intake and cancer is unclear at the present time [NAS 2001].

For this public health assessment, ATSDR derived exposure doses for Superior residents exposed to iron in the soil and water based on its pathway evaluation. Although iron absorption for dietary iron that the body obtains and uses from food is known, iron absorption from soil and water is not known. In general, iron that accumulates in soil should be less bioavailable than iron in the diet because any soluble iron in the soil would be expected to be washed out by rain, leaving behind the mostly insoluble iron compounds. However, as a conservative measure, ATSDR used an iron bioavailability factor of 100% when estimating site-specific iron exposure doses. In the following text, ATSDR provides its public health evaluation of site-specific iron exposures.

5.4.1 Soil

Residents are exposed to iron in sediment and waste tailings when they engage in hunting, fishing, wading, hiking, and other recreational activities on the mine site and along the floodplain area. Children's exposure doses based on the maximum iron level in sediment and waste tailings (4 mg/kg/day and 1 mg/kg/day, respectively) exceed EPA's oral RfD for iron (0.7 mg/kg/day); adult doses are below the RfD and therefore not of health concern. For a child ingesting 200 mg of sediment per day with the maximum iron concentration on the mine site (298,000 ppm), the dose would correspond to 60 mg of iron per day, which is above the tolerable upper intake level of 40 mg/day for children 7 months to 13 years old and equal to the dose where adults experienced gastrointestinal effects while taking iron supplements. The average iron concentration on the mine site (71,000 ppm) corresponds to a dose of 14 mg of iron per day, which is below the tolerable upper intake level. Exposure to iron found in the sediment and waste tailings in these areas would not be expected to pose a health hazard to children (1) because the body normally reduces absorption of iron from the gastrointestinal tract in response to higher concentrations, (2) because iron is not 100% bioavailable as assumed in ATSDR's dose calculations, and (3) because average iron concentrations are below known effect levels. Also, because the iron contaminated areas at the mine site and floodplain area are posted with warning signs, the frequency of recreational activities occurring in these areas is expected to be low.

In addition, residents are exposed to iron in waste tailings used as fill in the town of Superior. Prior to removal actions, a child's exposure dose based on the maximum levels of iron in both residential and non-residential soil (0.8 mg/kg/day) is comparable to EPA's oral RfD for iron (0.7 mg/kg/day) and therefore is not of health concern. Adult doses prior to removal actions, as

well as child and adult doses following removal actions, are below this RfD. As such, harmful health effects are not expected to result from past and current exposures to iron in town soil.

5.4.2 Surface Water

A past, current, and future completed exposure pathway to surface water exists for recreational users of Flat Creek. Iron was detected in 1 of 5 samples at a level of 96.5 ppb. Children's exposure doses based on this one detection of iron are not at a level of public health concern because the estimated child dose (0.0009 mg/kg/day) is several orders of magnitude below EPA's oral RfD. Likewise, harmful health effects are not expected for adults following exposure to iron in Flat Creek surface water while wading and fishing downstream of the mine site because the estimated exposure doses are several orders of magnitude below EPA's oral RfD.

In addition to recreational use, one family obtains part of their household water supply from a Flat Creek intake [EPA 2002a]. Iron was not detected in the one surface water sample collected from their Flat Creek intake. Daily ingestion of water containing the maximum iron level (96.5 ppb) detected in Flat Creek downstream of the mine site results in estimated child and adult doses that are two orders of magnitude below EPA's oral RfD. Based on these limited data, exposure to iron in the creek would not be expected to result in harmful health effects.

As stated previously, ATSDR consider Hall Gulch surface water an eliminated exposure pathway. Without exposure, adverse health effects are not expected. ATSDR recommends Hall Gulch continue to be posted with a warning sign informing the public that water coming out of the adit has levels of metals that may pose a health risk.

5.4.3 Groundwater

Past, current and future use of the three town of Superior wells and private wells are a completed exposure pathway. Past exposure to Flat Creek Spring is a completed exposure pathway. However, iron was detected only twice in the groundwater at levels of 210 ppb (private well) and 280 ppb (town well); iron was not detected in most samples. Daily ingestion of water containing these iron levels results in estimated child and adult doses that are one to two orders of magnitude below EPA's oral RfD. As such, harmful health effects are not expected to result from past and current exposures to iron in groundwater.

5.5 Lead

Lead is a naturally occurring bluish-gray metal found in the Earth's crust at about 15–20 mg/kg. Lead rarely occurs in its elemental state, but rather its +2 oxidation state in various ores throughout the earth. The most important lead containing ores are galena, anglesite, and cerussite. The largest industrial use of lead today is for the production of lead batteries, largely used in the automobile industry. Other uses of lead include the production of lead alloys, use in soldering materials, shielding for x-ray machines, and in the manufacture of corrosion and acid resistant materials used in the building industry. Today, lead can be found in all parts of our environment [ATSDR 2007b].

Although the use of lead as a gasoline additive has been gradually phased out and completely banned by 1995 in the United States and its use in paints was banned in 1978, human exposure to lead continues because lead does not degrade in the environment. Leaded paint is still prevalent in many older homes in the United States, and peeling or flaking paint contributes to indoor and outdoor dust levels.

Lead has been monitored in surface water, groundwater, and drinking water throughout the United States and other countries. The concentration of lead in surface water is highly variable depending upon sources of pollution, lead content of sediments, and characteristics of the system (like pH and temperature). The average lead level in surface water measured at 50,000 surface water stations throughout the United States is 3.9 ppb (based on 39,490 occurrences) [Eckel and Jacob 1988]. Levels of lead in surface water and groundwater throughout the United States typically range between 5 and 30 ppb, although levels as high as 890 ppb have been measured [EPA 1986b]. Lead levels in Flat Creek surface water ranged from 3.3 ppb to 233 ppb, with an average of about 24 ppb.

Based on several data sets, it is estimated that <1% of the public water systems in the United States have water entering the distribution system with lead levels above 5 ppb. These systems are estimated to serve <3% of the population that receives drinking water from public systems [EPA 1991]. For the town of Superior, lead levels in the distribution system ranged from 1 ppb to 10 ppb, with nine of the 10 detections below 5 ppb.

Local sources of lead releases can also contribute significantly to lead content in sediments [Gale et al. 2004]. For example, lead concentrations in sediments located near mines and or sites containing mine tailings in the old lead belt of Missouri were greatly elevated, 10,550–12,400 mg/kg sediment (dry weight) compared to unaffected sediments (72–400 mg/kg dry weight) [Gale et al. 2002]. The maximum lead concentration in Flat Creek sediments was 9,470 mg/kg.

Lead can affect almost every organ and system in the body, although the main target for lead toxicity is the nervous system. Children are more vulnerable to lead poisoning than adults. A child who swallows large amounts of lead may develop blood anemia, severe stomach-ache, muscle weakness, and brain damage. Unborn children can be exposed to lead through their mothers. Harmful health effects may include premature births, smaller babies, decreased mental ability, learning difficulties, and reduced growth in young children [ATSDR 2007b].

In general, the level of lead in a person's blood gives a good indication of recent exposure to lead and also correlates well with adverse health effects. Previously, CDC responded to the accumulated evidence of adverse effects associated with lead exposures by lowering the blood lead level (BLL) of concern from 60 µg/dL to 25 µg/dL. In 1991, CDC recommended lowering the level for individual intervention to 15 µg/dL and implementing community-wide primary lead poisoning prevention activities in areas where many children have BLLs greater than 10 µg/dL. However, this level, which was originally intended to trigger community-wide prevention activities, has been misinterpreted frequently as a definitive toxicologic threshold. There is growing evidence of adverse health effects in children with with blood lead levels below 10 µg/dL [CDC 2005].

Because there is no clear threshold for some of the more sensitive health effects, no guidelines for a safe dose of lead intake have been established. EPA has no RfD and ATSDR has no MRL to serve as a safe oral dose below which adverse health effects are unlikely to occur. However, lead cannot be entirely eliminated from the environment so there will always be some residual levels following cleanup actions at lead-contaminated sites.

Because neither ATSDR nor EPA have developed a MRL or RfD for exposure to lead, the usual approach of estimating exposure to an environmental contaminant and then comparing this dose to a health guideline cannot be used. Instead, environmental data are used to predict BLLs in

order to determine if any follow up action is needed. For this public health assessment, ATSDR used two approaches to predict BLLs.

1. ATSDR used an integrated regression analysis approach to evaluate lead exposures. This approach utilizes slope values from select studies that correlate environmental exposure to BLLs.
2. ATSDR evaluated exposure to lead by using a biological model that predicts a blood lead concentration that would result from exposure to environmental lead contamination. Specifically, ATSDR used EPA's Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) model.

For the regression analysis approach, environmental lead levels are multiplied by the percentage of time one is exposed to a particular source and then multiplied by an appropriate regression slope factor. The slope factors can be derived from regression analysis studies that determine BLLs for a similar route of exposure. Typically, these studies also identify standard errors describing the regression line of a particular source of lead exposure. These standard errors can be used to provide an upper and lower confidence limit contribution of each source of lead to BLLs. The individual source contributions can then be summed to provide an overall range estimate of BLLs [ATSDR 2007b].

For the biological model approach, the IEUBK model is designed to integrate exposure from lead in air, water, soil, dust, diet, paint, and other sources with pharmacokinetic modeling to predict blood lead concentrations in children 6 months to 7 years of age. The four main components of the current IEUBK model are: (1) an exposure model that relates environmental lead concentrations to age-dependent intake of lead into the gastrointestinal tract; (2) an absorption model that relates lead intake into the gastrointestinal tract and lead uptake into the blood; (3) a biokinetic model that relates lead uptake in the blood to the concentrations of lead in several organ and tissue compartments; and (4) a model for uncertainty in exposure and for population variability in absorption and biokinetics [EPA 1994].

The IEUBK model results can be viewed as a predictive tool for estimating changes in blood concentrations as exposures are modified [EPA 1994]. The IEUBK model provides choices a user may make in estimating a child's blood lead concentration. These are referred to "user-specified" parameters or decisions. The reliability of the results obtained using the model is very dependent on the selection of the various coefficients and default values that were used. In setting a lead cleanup level at a site, EPA's goal is to 'limit exposure to lead levels such that a typical child or group of similarly exposed children would have an estimated risk of no more than 5% of exceeding a blood lead level of 10 $\mu\text{g}/\text{dL}$ ' [EPA 1998].

In the following sections, ATSDR provides its evaluation of lead exposures using these two approaches.

5.5.1 Soil

As stated previously, a past, current, and future completed exposure pathway to surface soil, sediment, and waste tailings exists for people engaged in hunting, fishing, wading, hiking, and other recreational activities on the mine site and along the floodplain area. In addition, a past, current, and future completed exposure pathway exists to waste tailings used as fill in the town of Superior.

5.5.1.1 *Iron Mountain Mine and Mill Site and Flat Creek Floodplain*

Exposures at the mine site and along the floodplain area represent a short-term exposure scenario. EPA's IEUBK model is designed to evaluate chronic and relatively stable exposures from lead in environmental media [EPA 1994]. The reliability of the model for predicting BLLs for exposure durations shorter than 3 months has not been assessed. Using the model, even intermittent exposures (3 months), which are time-averaged over the entire year, may underestimate blood lead levels in children [EPA 2003b]. Because the model cannot assess short-term (acute) exposures, ATSDR used the slope factor regression analysis approach to evaluate mine site and floodplain area exposures.

For its slope factor analysis, ATSDR used the most protective correlation between BLLs and soil concentration found in the epidemiologic studies—a 0.0068- $\mu\text{g}/\text{dL}$ increase in blood lead level per ppm of lead in soil [Angle et al. 1984]. This correlation value, or slope factor, is based on studies where children (1–18 years old) were exposed to lead regularly and frequently in a residential setting. The standard errors describing the regression line for this particular study were 0.0068 ± 0.00097 . Using the average lead concentration measured in waste tailings (15,400 ppm), the slope factor analysis found that residents (especially children) exposed daily to these tailings could potentially increase their BLLs by 90–120 $\mu\text{g}/\text{dL}$. However, this estimated BLL range is likely overstated because the relationship between blood lead and environmental lead concentrations is somewhat non-linear at blood lead concentrations above 40 $\mu\text{g}/\text{dL}$ in adults and 30 $\mu\text{g}/\text{dL}$ in children [EPA 1994]. The average lead concentration measured in sediment affected by waste tailings (2,500 ppm) could potentially increase BLLs by 15–19 $\mu\text{g}/\text{dL}$. These ranges are above CDC's level of concern (10 $\mu\text{g}/\text{dL}$) and therefore would be of health concern if residents, particularly children, visited these areas and engaged in contact-intensive activities on a daily basis.

As stated previously, because the contaminated areas at the mine site and Flat Creek floodplain area are posted with warning signs, ATSDR would expect the frequency of recreational activities occurring in these areas to be low. However, if people are visiting these areas, ATSDR recommends they take prudent public health measures to reduce their ingestion or inhalation of dust and soil.

5.5.1.2 *Town of Superior*

The use of solely default parameters in the IEUBK model yields a soil lead level of about 400 ppm, which EPA recommends as a soil screening level (SSL) for lead in soil at residential properties [EPA 1998]. Removal actions occurred at five residential yards. One of these yards contained soil lead levels of 757 ppm and 1,770 ppm. The other four yards' soil lead levels were 1,620 ppm, 4,000 ppm, 8,530 ppm and 16,700 ppm. For the slope factor regression approach, using the most protective slope factor (0.0068 ± 0.00097), daily exposure to the average soil lead level from the five residential yards where removal efforts occurred (5,563 ppm) could have potentially increased BLLs by 32–43 $\mu\text{g}/\text{dL}$. ATSDR was not able to use EPA's IEUBK model for the residential yard scenario because the average lead concentration (5,563 ppm) is associated with blood lead levels above 30 $\mu\text{g}/\text{dL}$, which is above the range of values that were used in the calibration and empirical validation of the model.

Prior to removal actions, several non-residential areas of town, including the high school track, had the potential for elevating BLLs above a level of concern. For example, the average soil lead level at the high school track (3,806 ppm) could have potentially increased children's BLLs by

22–30 µg/dL with daily exposure. Using EPA’s IEUBK model with default parameters for all inputs except soil, the probability is high (approximately 83%) that a child exposed to soil at the high school track in the past could have had a BLL greater than 10 µg/dL assuming daily exposure.

Therefore, ATSDR considers residents’ (especially children’s) daily exposure in the past to soil in these areas of town with elevated lead concentrations to have been of public health concern. Because the 2002 TCRA removed contaminated soil from these areas, current and future exposures to soil lead are not expected to be at levels of health concern in these areas. However, some uncertainty exists about the levels of lead that remain. Additional characterization (e.g., post-confirmation sampling and analysis) of these areas would be needed to ensure that removal efforts were successful.

ATSDR also evaluated current and future exposures to lead in soil for those areas in Superior that were sampled but not included in the 2002 removal action. Because current soil data are not available, ATSDR *estimated* potential current contamination levels using data from the 2002 sampling event to provide some perspective on the potential that soil lead contamination in Superior may currently be of health concern in some areas. Based on these estimates, most residential and non-residential areas contained soil lead levels below 400 ppm. Therefore, ATSDR finds that exposures to estimated current lead levels for most residential and non-residential areas are not at levels of public health concern, with exceptions noted in the following text.

ATSDR’s evaluation assumed the 2002 sampling event was successful in targeting and sampling those properties of potential concern. Because the sampling effort was dependent on residents identifying where fill material existed, it is not possible for ATSDR to determine whether the effort identified all town locations with waste tailings material. Therefore, the potential exists that additional properties in town that were not sampled could still contain waste tailings material with levels of lead of public health concern. ATSDR’s finds that additional efforts, such as another mail-out to town residents or a town hall meeting, may be beneficial in determining whether additional areas of town should be characterized.

In addition, following removal actions, according to ATSDR’s subset of laboratory data, there are four yards with soil lead levels above 400 ppm. The average lead level of the four yard samples is 982 ppm, with a range of 523 ppm to 1,680 ppm. Two of these four residences also contain elevated arsenic levels.

For the slope factor regression approach, using the most protective slope factor (0.0068 ± 0.00097), daily exposure to the average soil lead level of these four residential yards could potentially increase children’s BLLs by 6–8 µg/dL. Using default parameters for all inputs except soil, the IEUBK model predicts that a child (or a similarly exposed group of children) would have a 19% probability of exceeding a BLL of 10 µg/dL if exposed daily to 982 ppm lead in soil. If the interior dust lead level is assumed to be 45% of the yard soil lead level, the probability for a child or similarly exposed group of children to exceed a level of concern is 41% at these four residences. Overall, ATSDR’s evaluation indicates that the potential exists for children to have BLLs of health concern following daily exposure to the maximum lead levels at these four residences.

At this time, it is not known whether the one sample collected from each of the four yards is representative of the average lead level that children might be exposed to in each of these four

yards. Additional characterization of these four yards is needed to determine whether the detected lead levels are truly representative.

Following removal actions, for the remaining non-residential soil samples, four samples were above the residential lead SSL. The average lead concentration in these four non-residential soil samples was 561 ppm, with a range of 433 ppm to 689 ppm. The IEUBK model predicts that a child (or a similarly exposed group of children) would have a 6% probability of exceeding a BLL of 10 µg/dL if exposed daily to 561 ppm lead in soil (with all model default inputs except soil). For the slope factor regression approach, using the most protective slope factor (0.0068 ± 0.00097), daily exposure to the average soil lead level in these non-residential areas could potentially increase children's BLLs by approximately 3–4 µg/dL. Of note, it is unlikely that children are exposed daily to these non-residential areas. Therefore, ATSDR finds that harmful health effects are unlikely considering the lead levels detected in these non-residential areas.

Overall, when deriving a site-specific soil cleanup level for lead, EPA considers aspects such as site-specific variability in exposure, lead geochemistry, and projected land use. EPA can also factor in other considerations such as cost, technical feasibility, compliance with state and federal regulations, and community acceptance. These factors result in large variations in proposed cleanup levels at different lead-contaminated sites. To prioritize response actions, EPA supplies guidance for interpreting soil sample results at residential lead sites in their Superfund Lead-Contaminated Residential Sites Handbook [EPA 2003c]. For sites with sensitive populations (like young children) and soil lead concentrations in surface soils between 400 ppm and 1,200 ppm, properties can be addressed through responses such as non-time-critical removal actions (NTCRAs) or long-term remedial actions. ATSDR is aware that additional response actions are being considered. In the interim, ATSDR recommends residents of these four residential yards take prudent public health measures to reduce their exposure. In addition, any residents who suspect waste tailings fill material to be in their yards should also take action to reduce exposure to lead contamination (see Appendix D).

5.5.2 Surface Water

A past, current, and future completed exposure pathway to surface water exists for children and adults following exposure to Flat Creek water. Because the IEUBK model cannot assess short-term (acute exposures), ATSDR used the slope factor regression analysis approach.

For its slope factor analysis, ATSDR used the most protective slope factor between BLLs and drinking water concentration found in the epidemiologic studies—a 0.26-µg/dL increase in blood lead level per ppb of lead in water [Lacey et al. 1985]. This correlation value, or slope factor, is based on a study where infants were exposed to lead in their bottles (formula mixed with water) at concentrations greater than 15 ppb. Daily ingestion exposure to the average surface water lead levels (about 24 ppb) could potentially increase BLLs by about 6 µg/dL. However, exposure is expected to be intermittent and visitors (hunters, hikers, etc.) are not expected to be frequently drinking the water. Therefore, harmful health effects are not expected for visitors who incidentally ingest Flat Creek surface water.

As stated previously, one homeowner receives part of their drinking water from a Flat Creek intake. Total lead was not detected in the one sample from their intake, although dissolved lead was detected at 1 ppb. Although this lead level is not of health concern, according to the limited data available, average lead levels in Flat Creek are about 24 ppb and range from not detected to

233 ppb. These lead levels exceed EPA's action level for lead in drinking water (15 ppb). For the slope factor approach, daily exposure to the average surface water lead levels (about 24 ppb) could potentially increase BLLs by about 6 $\mu\text{g}/\text{dL}$. The IEUBK model predicts that a child (or a similarly exposed group of children) would have a 3.4% probability of exceeding a BLL of 10 $\mu\text{g}/\text{dL}$ if exposed daily to 24 ppb lead in drinking water (with all model default inputs except water lead concentration). When an intake rate of 1 liter is used instead of the default rates, the IEUBK model predicts a 15% probability exceeding a BLL of 10 $\mu\text{g}/\text{dL}$ if exposed daily to 24 ppb lead in drinking water. ATSDR's evaluation indicates that the potential exists for children who ingest 1 liter of Flat Creek water per day to have BLLs of health concern. In general, ATSDR considers efforts to reduce drinking water exposures a prudent public health measure when chemical levels are above regulatory guidelines. Of note, this home also receives their drinking water from a private well. If the homeowners are concerned about potential lead exposures via their Flat Creek intake, ATSDR would recommend they use their private well water as their drinking water source.

As stated previously, because shallow, reddish-brown surface water exists in Hall Gulch, ATSDR would not expect people to occasionally drink, or wade in, this surface water. Because lead levels would be of public health concern if the water were to be frequently ingested, ATSDR recommends Hall Gulch continue to be posted with a warning sign informing the public that water coming out of the adit has levels of metals that may pose a health risk.

5.5.3 Groundwater

Lead was detected in the distribution system of the current public water supply in 10 of 13 samples, with an average of 3 ppb. Lead was not detected in the seven samples collected from Wells 1, 2 and 3. Also, lead was not detected from the one sample collected from Flat Creek Spring nor the one sample collected from a private well. For the IEUBK model approach, the default lead concentration used in the model for water is 4 ppb. Because the distribution system data indicate an average below this value, exposure to these lead levels would have an estimated risk of less than 5% of exceeding a blood lead level of 10 $\mu\text{g}/\text{dL}$.

For its slope factor analysis, ATSDR used the most protective slope factor between BLLs and drinking water concentration found in the epidemiologic studies (0.26- $\mu\text{g}/\text{dL}$). Using the average lead concentration measured in distribution system water (3 ppb), residents (especially infants) exposed daily could increase their BLLs less than 1 $\mu\text{g}/\text{dL}$.

Also of note, the lead levels in the distribution system samples were below EPA's action level of 15 ppb. Overall, although the data are limited, lead in groundwater does not appear to be at levels of public health concern.

5.5.4 Total Lead Exposure

In February 2002, there were 66 Superior residents who were tested for lead exposure. As stated previously, all blood lead concentrations were below 10 $\mu\text{g}/\text{dL}$. However, these blood lead results do not represent peak exposure levels to soil, such as those that might occur in the summer when outdoor activities occur.

For its assessment, ATSDR did not sum up the various contributions to total blood lead from each medium because including short-term exposures from the mine site and floodplain area, as well as for four residential yards, would automatically result in BLLs above 10 $\mu\text{g}/\text{dL}$.

5.6 Manganese

Manganese is naturally found in many types of rocks and comprises about 0.1% of the earth's crust [Graedel 1978, NAS 1973]. Pure manganese is a silver-colored metal; it does not, however, occur in the environment as a pure metal. Manganese does not have a distinctive taste or smell. It usually combines with other elements (e.g., oxygen, sulfur, and chlorine) to form compounds. Manganese can change from one compound to another through natural processes or by human activity, but it does not break down or disappear from the environment. Manganese compounds are mined and used to produce manganese metal, which is combined with iron to make various types of steel. Some manganese compounds are used in the production of batteries, in dietary supplements, and as ingredients in ceramics, pesticides, and fertilizers. Additionally, manganese is present in many foods, including grains and cereals, and is found in high concentrations in tea [ATSDR 2008c].

Dissolved manganese concentrations in United States surface waters averaged 59 ppb in 1962–1967 and 24 ppb in 1974–1981 [Kopp and Kroner 1967, Smith et al. 1987]. Mean manganese concentrations in groundwater are similar to those in surface water [ATSDR 2008c]. Several studies report mean manganese concentrations in public drinking water supplies ranging from 4 to 32 ppb [DHEW 1970, EPA 1984, NAS 1980, WHO 1981].

Average background levels of manganese in soils range from 40–900 ppm, with an estimated average concentration of 330 ppm [Cooper 1984, Eckel and Langley 1988, EPA 1985b, Rope et al. 1988, Schroeder et al. 1987]. Maximum concentrations of manganese were detected in sediment along Flat Creek at concentrations of 66,100 ppm and in soil at the fairgrounds in Superior at 4,250 ppm.

Manganese is an essential trace element and is required by the body to break down amino acids and produce energy. Therefore, people typically have small amounts of manganese in their bodies. Most people consume about 2–5 mg/day of manganese in their diets [EPA 2008]. Too little manganese in one's diet can cause slowed blood clotting, skin problems, changes in hair color, lowered cholesterol levels, and other metabolic alterations [ATSDR 2008c].

Manganese can enter the body via ingestion, but most manganese is excreted in feces—only 3–5% of manganese is absorbed by the body when ingested [Davidsson et al. 1988, Mena et al. 1969]. Under normal circumstances, the amount of manganese is regulated so the body has neither too much nor too little [EPA 1984]. For example, if large amounts of manganese are consumed, large amounts will be excreted. The total amount of manganese in the body tends to stay the same even when exposed to higher levels than usual. Still, if too much manganese is ingested, the body might not be able to adjust and eliminate the additional amount.

The Food and Nutrition Board of the National Research Council determined that 2–5 mg/day of manganese is an “estimated safe and adequate daily dietary intake” for adults [NRC 1989]. The World Health Organization (WHO) concluded that 2–3 mg/day is “adequate” for adults and 8–9 mg/day is “perfectly safe” [WHO 1973]. Another study determined that 3.5–7 mg/day is a safe and adequate level for adults [Freeland-Graves et al. 1987]. The National Health and Medical Research Council nutrient reference values for Australia and New Zealand state the adequate intake for children aged 1–18 years range from 2.0–3.5 mg/day [NHMRC 2009].

EPA determined that an appropriate RfD for manganese in the diet is 0.14 mg/kg/day; however, EPA recommends that a modifying factor of 3 be applied when assessing risk from manganese in

drinking water or soil, resulting in an environmental media RfD of 0.05 mg/kg/day. EPA also reports a NOAEL of 10 mg/day (or 0.14 mg/kg-day for 70 kg adult) for chronic human consumption of manganese in the diet, which is based on a composite of data from several studies [EPA 2008]. ATSDR has not derived an MRL from oral exposure to manganese. NAS calculated a tolerable upper intake level of 11 mg/day for adults based on a NOAEL for Western diets [NAS 2001].

EPA has determined that manganese is not classifiable as to human carcinogenicity because existing studies are inadequate [EPA 2008]. DHHS has also not classified manganese as to its carcinogenicity. However, there is little evidence to indicate that cancer is a major concern for people exposed to manganese in the environment [ATSDR 2008c].

For this health consultation, ATSDR derived exposure doses for Superior residents exposed to manganese in the soil and water based on its pathway evaluation. In the following text, ATSDR provides a description of its public health evaluation of manganese exposures.

5.6.1 Soil

Residents are exposed to manganese in sediment and waste tailings when they engage in hunting, fishing, wading, hiking, and other recreational activities on the mine site and along the floodplain area. For the short-term exposure scenario, children's exposure doses based on the maximum manganese level in sediment and waste tailings are 0.8 mg/kg/day and 0.07 mg/kg/day, respectively. Both estimated manganese exposure doses are above EPA's RfD for environmental media (0.05 mg/kg/day). As noted previously, EPA's RfDs are estimates of daily exposures to a substance that are likely to be without a discernable risk of deleterious effects to the general human population (including sensitive subgroups) during a *lifetime* of exposure. Children's estimated doses to average manganese levels in sediment and waste tailings are comparable to or below EPA's RfD for environmental media. Adult doses are lower still. Exposure to manganese found in the sediment and waste tailings in these areas would not be expected to be of health concern because (1) a slight increase in manganese intake would most likely lead to a slight increase in the amount that is excreted, (2) because manganese is not 100% bioavailable as assumed in ATSDR's dose calculations, and (3) because exposure to average manganese levels in sediment and waste tailings results in estimated doses that are at or below EPA's RfD. Also, because the manganese contaminated areas at the mine site and floodplain area are posted with warning signs, the frequency of recreational activities occurring in these areas is expected to be low.

In addition, residents are exposed to manganese in waste tailings used as fill in the town of Superior. Prior to removal actions, children's exposure doses based on the maximum levels of manganese in soil at the fairgrounds (0.05 mg/kg/day) and a residence (0.02 mg/kg/day) are comparable to EPA's RfD (0.05 mg/kg/day) for environmental media. Following removal actions, a child's exposure dose based on the maximum levels of manganese in residential soil (0.01 mg/kg/day) is comparable to this RfD as well. The exposure doses for adults are lower still. Because manganese in soil is not 100% bioavailable as assumed in ATSDR's dose calculations and because soil manganese concentrations are at or below EPA's RfD, harmful health effects are not expected to result from past and current exposure to manganese in town soil.

5.6.2 Surface Water

A past, current, and future completed exposure pathway to surface water exists for recreational users of Flat Creek and for the one family who obtains part of their household water supply from a Flat Creek intake. However, manganese has not been detected in creek water above ATSDR's health-based comparison values. Therefore, harmful health effects are not expected for children and adults (1) following exposure to manganese in Flat Creek surface water while wading and fishing downstream from the mine site and (2) following exposure to manganese from drinking, showering, bathing, cooking, and washing dishes with this surface water.

In the Hall Gulch mine adit area, manganese was detected in 7 of 9 surface water samples above a health-based comparison value. As stated previously, ATSDR considers Hall Gulch surface water an eliminated exposure pathway because the agency does not expect people to occasionally drink, or wade in, this shallow, reddish-brown surface water. Without exposure, adverse health effects are not expected.

5.6.3 Groundwater

Manganese has not been detected above health-based comparison values in the limited number of samples collected from one private well and from Flat Creek Spring. ATSDR does not have data to suggest that the public water supply contains manganese at levels of health concern either as it has not been detected. As such, harmful health effects are not expected.

5.7 Thallium

Thallium is naturally and widely distributed in the Earth's crust. Pure thallium is a soft, bluish-white metal that is odorless and tasteless. In the environment, it can be found in pure form, mixed with other metals to form alloys, or combined with other substances (e.g., bromine, chlorine, fluorine, and iodine) to form salts. Thallium cannot be broken down to simpler substances. It exists in two chemical states—thallous (monovalent) and thallic (trivalent). The thallous state is more common and more stable, and the form to which people are more likely to be exposed in the environment [ATSDR 1992b].

At one point, thallium was used therapeutically to cure various infections. It was also used in the past as a rodenticide. However, both uses of thallium stopped because of its toxicity to man [ATSDR 1992b, IPCS 1996]. Currently, thallium is used in low temperature thermometers, special glasses with a high resistance and a low melting point, mixed crystals for infrared instruments, electronic devices, and mercury lamps. Thallium is no longer produced in the United States [ATSDR 1992b, IPCS 1996].

In a survey of tap water from 3,834 homes in the United States, an average concentration of 0.89 ppb of thallium was detected [EPA 1980a, EPA 1988]. Concentrations in rivers that receive mining operation effluents in the United States and Canada ranged from 0.7 to 88.3 ppb [EPA 1980a, EPA 1988, Zitko et al. 1975]. Total thallium concentrations in soil typically range from 0.1–1.0 ppm [Bowen 1966, Bowen 1979, Brumsack 1977, Chattopadhyay and Jervis 1974, Schoer 1984, Smith and Carson 1977]. However, higher concentrations can be found in organic-rich shales (25 ppm) and near some metallic ore deposits (up to 5 ppm) [Smith and Carson 1977]. Maximum concentrations of thallium were detected in sediment near the mine adit at 11.5 ppm, in waste tailings along Flat Creek at concentrations of 12.6 ppm, and at the fairgrounds in Superior at 6.4 ppm.

Although no quantitative data exist, thallium is assumed to be absorbed through the skin [ATSDR 1992b, IPCS 1996]. Limited data about the absorption of thallium from the gastrointestinal tract in humans exist [ATSDR 1992b, IPCS 1996]. The results from one study suggest that most ingested thallium is absorbed [Barclay et al. 1953]. Once absorbed, thallium is rapidly distributed throughout the body, with the kidney accumulating the highest concentrations. In humans, thallium is eliminated mainly through urine and to a lesser extent through feces. Approximately half of the thallium entering the human body will exit within 3–10 days, however, a biological half-life of 30 days has been reported in humans [ATSDR 1992b, IPCS 1996].

Limited human data are available on health effects associated with thallium exposure. EPA derived an oral RfD for thallium (soluble salts) of 0.000065 mg/kg/day. EPA has also established RfDs of 0.00008 and 0.00009 mg/kg/day for several thallium compounds (thallium acetate, thallium carbonate, thallium chloride, thallium nitrate, and thallium sulfate). These RfDs are based on a failure to observe harmful effects in rats that were administered as much as 0.25 mg/kg/day (NOAEL) of thallium sulfate by gavage (stomach tube) for 90 days [EPA 1986a].

ATSDR has not derived an MRL for ingestion of thallium, but reports a less serious LOAEL following intermediate oral exposure to thallium based on hair loss—shown to be a temporary effect—for doses ranging from 1.2–1.8 mg/kg/day [Downs et al. 1960]. The International Programme on Chemical Safety (IPCS) determined that a daily intake of 10 micrograms per day ($\mu\text{g}/\text{day}$) of a soluble thallium compound is unlikely to cause harmful health effects [IPCS 1996].

ATSDR derived exposure doses for Superior residents exposed to thallium in the soil and water based on its pathway evaluation. In the following text, ATSDR provides a description of its public health evaluation of thallium exposures.

5.7.1 Soil

Residents are exposed to thallium in sediment and waste tailings when they engage in hunting, fishing, wading, hiking, and other recreational activities on the mill site and along the floodplain area. For the short-term exposure scenario, children's exposure doses based on the maximum thallium level in sediment and waste tailings are not at a level of public health concern because the estimated child doses (0.0001 mg/kg/day and 0.0002 mg/kg/day, respectively) are several orders of magnitude below the NOAEL (0.25 mg/kg/day) and the LOAEL dose range (1.2–1.8 mg/kg/day) reported for intermediate exposures (15 days to less than a year). Adult exposures to the maximum thallium levels in sediment and waste tailings, as well as child and adult exposures to average thallium levels in these media, are not at a level of health concern because the estimated doses are also several orders of magnitude below thallium's NOAEL and lowest LOAEL dose range. Therefore, for the short-term exposure scenario, harmful health effects are not expected to result from exposure to thallium in sediment and waste tailings from the mill site and floodplain area.

Thallium was not detected in most soil samples collected within the town of Superior. Prior to removal actions, thallium exceeded its health-based comparison value in only one sample. A child's exposure dose based on this sample, which was collected at the fairgrounds, is not at a level of health concern because the estimated dose (0.00008 mg/kg/day) is comparable to EPA's RfDs and several orders of magnitude below the NOAEL (0.25 mg/kg/day). The exposure dose for adults is lower still. As such, harmful health effects are not expected to result from past and current exposure to thallium in town soil.

5.7.2 *Surface Water*

A past, current, and future completed exposure pathway to surface water exists for recreational users of Flat Creek and for the one family who obtains part of their household water supply from a Flat Creek intake. In creek water, thallium was detected at levels below EPA's MCL; most samples were non-detect for thallium. As stated previously, MCLs are considered protective of public health over a lifetime of exposure. Therefore, harmful health effects from thallium are not expected for children and adults (1) following exposure to Flat Creek surface water while wading and fishing downstream from the mine site and (2) following exposure from drinking, showering, bathing, cooking, and washing dishes with this surface water.

5.7.3 *Groundwater*

Past, current and future use of the three town of Superior wells and private wells are a completed exposure pathway. Past exposure to Flat Creek Spring is a completed exposure pathway. However, thallium has not been detected above health-based comparison values in the limited number of samples collected from the one private well and from Flat Creek Spring. ATSDR does not have data to suggest that the public water supply contains thallium at levels of health concern either as it has not been detected. As such, harmful health effects are not expected to result from past, current, and future exposures to thallium in groundwater.

5.8 **Zinc**

Zinc is one of the most common elements in the Earth's crust. Metallic zinc is a bluish-white shiny metal and is often mixed with other metals to form alloys such as brass and bronze (e.g., pennies are made out of a zinc-copper alloy). A common use for zinc is to coat steel and iron to prevent rust and corrosion, a process called galvanization. Zinc can also combine with other elements, such as chlorine (e.g., zinc chloride), oxygen (e.g., zinc oxide), and sulfur (e.g., zinc sulfide), to form compounds [ATSDR 2005a].

Zinc sulfide is the most common zinc ore found naturally in the environment. Zinc compounds are used in a variety of industries—zinc sulfide and zinc oxide are used to make white paints and ceramics; zinc oxide is used to produce rubber; and zinc acetate, zinc chloride, and zinc sulfate are used in preserving wood and in manufacturing and dyeing fabrics. Zinc compounds are also widely used as ingredients in vitamin supplements; sun blocks; diaper rash ointments; deodorants; athlete's foot, acne, and poison ivy preparations; and antidandruff shampoos [ATSDR 2005a].

Background zinc concentrations in surface water are usually less than 50 ppb; however, concentrations in surface water and groundwater can range from 2.0–50,000 ppb [EPA 1980b, NAS 1977]. Higher concentrations are common in many locations and appear to be correlated with mining activities and/or geological areas rich in zinc [EPA 1980b]. Concentrations of zinc in drinking water can be higher than levels in surface water due to treatment of the distribution system and contamination from the plumbing [NAS 1977]. However, the available data suggest that zinc concentrations in drinking water are still much lower than the recommended daily intake levels [ATSDR 2005a].

Background concentrations of zinc ranged from less than 5 to 2,900 ppm in soil samples collected from 1,318 uncontaminated areas across the conterminous United States from 1961 through the autumn of 1975. The average concentration was 60 ppm [Shacklette and Boerngen 1984]. The mean concentrations of zinc vary according to land use—the average is 25 ppm in

agricultural areas, 75 ppm in suburban residential areas, 157 ppm in mixed industrial/residential areas, and 360 ppm in industrial inner urban areas [Haines 1984]. Maximum concentrations of zinc were detected in sediment near the mine adit (75,900 ppm) and at the fairgrounds in Superior (7,300 ppm).

Zinc is an essential nutrient that is needed by the body for catalytic (e.g., nearly 100 enzymes depend on zinc), structural (e.g., proteins form domains that facilitate the production of biologically active molecules), and regulatory functions (e.g., metallothionein expression) [Cousins 1996]. Zinc deficiency has been associated with dermatitis, anorexia, growth retardation, poor wound healing, reproductive problems, a lowered resistance to disease, and depressed mental function [ATSDR 2005a]. The NAS RDA is 8 mg/day for adult women and 11 mg/day for adult men; these doses correspond to approximately 0.13 mg/kg/day for women and 0.16 mg/kg/day for men. A daily intake of 12 mg/day is recommended for pregnant and nursing women [ATSDR 2005a; NAS 2001]. NAS calculated a tolerable upper intake level of 40 mg/day for adults, based on reduction in erythrocyte copper-zinc superoxide dismutase activity [NAS 2001].

The absorption and metabolism of ingested zinc has been well studied. Several studies found varying absorption (ranging from 8 to 81%) due to a number of factors, including the solubility of the zinc compound ingested, the amount of zinc ingested, the amount and kind of food eaten, inhibitors (e.g., calcium, phosphorus, and dietary fiber and phytates), and enhancers (e.g., amino acids, picolinic acid, and prostaglandin E2) [Aamodt et al. 1983, Hunt et al. 1991, Istfan et al. 1983, Reinhold et al. 1991, Sandstrom and Abrahamson 1989, Sandstrom and Cederblad 1980, Sandstrom and Sandberg 1992]. The body uses a homeostatic mechanism to control zinc absorption in the gastrointestinal tract [Davies 1980]. People with adequate nutritional levels of zinc tend to absorb 20–30% of ingested zinc, whereas people with zinc deficiencies absorb more [Johnson et al. 1988, Spencer et al. 1985]. Once absorbed, zinc is widely distributed throughout the body, and is excreted in both urine and feces. The bioavailability of zinc ingested in contaminated water and soil has not been estimated. Although, we do know that the bioavailability is higher in media with a low pH due to increased zinc solubility and ionization [ATSDR 2005a].

ATSDR's intermediate and chronic oral MRL for zinc (0.3 mg/kg/day) is based on a study in which subclinical hematological health effects were observed when 18 healthy women were given doses of 0.83 mg/kg/day of a zinc supplement for 10 weeks [Yadrick et al. 1989]. Because the observed effect is considered to be a precursor event to more severe symptoms, rather than a toxic effect of itself, 0.83 mg/kg/day was considered to be a NOAEL. The MRL was calculated based on the assumption of healthy dietary levels and represents exposure beyond the normal diet that is believed to be without an appreciable risk of toxic response [ATSDR 2005a]. The derivation of the MRL is supported by several other studies that investigated effects from zinc supplementation in humans [Black et al. 1988, Davis et al. 2000, Fischer et al. 1984, Freeland-Graves et al. 1982, Milne et al. 2001, Prasad et al. 1978].

EPA derived the same value for an oral RfD for zinc (0.3 mg/kg/day). However, EPA calculated a total dose by adding the average daily intake levels from FDA's 1982–1986 Total Diet Study [Pennington et al. 1989] to the reported supplemental doses from four principal studies. All four studies identified physiological changes in similar, sensitive endpoints at similar dose levels in a variety of human subject groups. These studies identified effect levels of 0.81 mg/kg/day for postmenopausal women [Davis et al. 2000, Milne et al. 2001], 0.94 mg/kg/day in healthy adult

males [Fischer et al. 1984], and 0.99 mg/kg/day in healthy adult women [Yadrick et al. 1989]. The effect levels from these four studies were averaged together to obtain a LOAEL of 0.91 mg/kg/day [EPA 2008].

There are no reports on the carcinogenicity of zinc in humans. EPA has determined that zinc is not classifiable as to human carcinogenicity because studies of humans occupationally exposed to zinc are inadequate or inconclusive, adequate animal bioassays of the possible carcinogenicity of zinc are not available, and results of genotoxic tests of zinc have been equivocal [EPA 2008]. DHHS has also not classified zinc as to its carcinogenicity.

For this health consultation, ATSDR derived exposure doses for Superior residents exposed to zinc in the soil and water based on its pathway evaluation. In the following text, ATSDR provides its public health evaluation of zinc exposures in Superior.

5.8.1 Soil

Residents are exposed to zinc in sediment and waste tailings when they engage in hunting, fishing, wading, hiking, and other recreational activities on the mill site and along the floodplain area. For the short-term exposure scenario, children's exposure doses based on the maximum zinc level in sediment and waste tailings are 0.9 mg/kg/day and 0.3 mg/kg/day, respectively. These estimated zinc exposure doses are comparable with ATSDR's reported NOAEL (0.83 mg/kg/day) and EPA's reported LOAEL range (0.81–0.99 mg/kg/day). Adult exposures to the maximum zinc levels in sediment and waste tailings, as well as child and adult exposures to average zinc levels in these media, are below ATSDR's NOAEL and EPA's LOAEL dose range. Because (1) the human body uses a homeostatic mechanism to control zinc absorption, (2) zinc in soil is not 100% bioavailable as assumed in ATSDR's dose calculations, and (3) the average soil zinc concentrations are below known effect levels, ATSDR would not expect short-term zinc exposures to result in harmful health effects in children or adults who visit this area. In addition, the frequency of recreational activities occurring in these areas is expected to be low.

Prior to and following removal efforts, zinc was detected below health-based comparison values in soil samples collected within the town of Superior. As such, harmful health effects are not expected to result from past and current exposure to zinc in town soil.

5.8.2 Surface Water

A past, current, and future completed exposure pathway to surface water exists for recreational users of Flat Creek and for the one family who obtains part of their household water supply from a Flat Creek intake. However, zinc was detected below ATSDR's health-based comparison values. As such, harmful health effects are not expected.

Of note, elevated levels of zinc were detected surface water in the Hall Gulch mine adit area. As stated previously, ATSDR consider Hall Gulch surface water an eliminated exposure pathway. Without exposure, adverse health effects are not expected. ATSDR recommends Hall Gulch continue to be posted with a warning sign informing the public that water coming out of the adit has levels of metals that may pose a health risk.

5.8.3 Groundwater

Data are not available for zinc in the distribution system. However, limited groundwater samples from a private well and the Flat Creek Spring indicate levels of zinc below ATSDR's health-

based comparison values. As such, harmful health effects are not expected to result from past, current, and future exposures to zinc in groundwater.

5.9 Limitations

ATSDR's public health evaluation has several limitations, some of which are noted in this section. These limitations include, but are not limited to, the following:

- For some of the reports reviewed, the data analyses appear to have been conducted in accordance with standard data quality practices, but no documentation was available to confirm the level of QA/QC. For its evaluation, ATSDR assumed all data were suitable for public health evaluation purposes.
- For the mine site and floodplain area, no soil samples were collected. Samples were collected directly from waste tailings material and from sediment affected by waste tailings. Although contact with these waste tailings deposits are of public health concern, ATSDR cannot provide a definitive conclusion regarding the health risks associated with exposures to soil that do not appear to be affected by waste tailings because representative data for these areas were not available.
- ASARCO conducted reclamation activities that included re-vegetation of some Flat Creek tailings and removal of other tailings from Flat Creek to an impoundment on ASARCO property [EPA 2002d]. However, there are no post-remediation sampling data available to confirm the success or extent of these remediation activities.
- In 2002, EPA conducted a TCRA in the town of Superior. Although contaminated soil was removed from public and private properties, only limited soil data from the excavation effort are available to characterize the removal activities.
- Several residents on the north side of town rely on private wells to obtain household water. However, few details are documented about these wells. With the exception of one well, no environmental data are available for these wells.
- Estimating an exposure dose required identifying how much, how often, and how long a person may come in contact with some concentration of the contaminant in the water and soil. ATSDR made several assumptions with regard to site-specific exposure scenarios. Although ATSDR's assumptions were conservative, each person's exposure may either increase or decrease depending on their lifestyle and individual characteristics that influence contact with contaminated media.
- Estimating exposure doses also required identifying characteristics about the specific chemicals of concern. When site-specific data were unavailable, ATSDR made conservative assumptions. For example, ATSDR assumed the bioavailability of most chemicals was 100% in the exposure dose calculations even though the likelihood is that the bioavailability is less than 100%.
- ATSDR's evaluation required the examination and interpretation of reliable substance-specific health effects data. This included a review of epidemiologic (human) and experimental (animal) studies. Clearly, a study based on human data would hold the greatest weight in describing relationships between a particular exposure and a human health effect but in many cases, only experimental studies were available. In addition,

the number of studies available for a chemical may not be sufficient to provide a clear picture of the true dose-response, especially where the exposure is to low doses.

- The two approaches used in ATSDR's lead evaluation, the slope factor analysis and the IEUBK model, have some limitations. For example, a limitation of the slope factor approach is that it assumes a linear relationship between BLLs and the uptake of lead from environmental media; however, elevated BLLs (>30 µg/L) typically exhibit a non-linear relationship. An example of a limitation of the IEUBK model is that the model was designed to evaluate relatively stable exposure situations, rather than rapidly varying exposures or short-term exposures. The IEUBK model was also not developed to assess lead risks for age groups older than 7 years.

Overall, ATSDR recognizes there are uncertainties in its public health evaluation. However, providing a framework that puts site-specific exposures and the potential for harm into perspective is one of the primary goals of ATSDR's public health evaluation process (ATSDR 2005b).

6 Child Health Considerations

In communities faced with environmental contamination, the many physical differences between children and adults need to be emphasized. Children could be at greater risk than adults from certain kinds of exposure to hazardous substances. Children play outdoors and sometimes engage in hand-to-mouth behaviors that increase their exposure potential. Children are shorter than are adults, and thus they breathe dust, soil, and vapors closer to the ground. A child's lower body weight and higher air intake rate results in a greater dose of hazardous substances per unit of body weight. If toxic exposure levels are high enough during critical growth stages, the developing body systems of children can sustain permanent damage. Finally, children are dependent on adults for access to housing, medical care, and risk identification. Thus, adults need as much information as possible to make informed decisions about their children's health. Therefore, in this public health assessment, ATSDR's has particularly focused on the evaluation of children's exposure to heavy metal contamination and on the potential health effects associated with these exposures. Please refer to Section 5, Public Health Evaluation, which contains detailed discussions on children's exposures.

Of note, sensitive populations not discussed specifically in this public health assessment, such as children who eat non-food items like soil (that is, exhibit pica behavior), could also receive doses of health concern. Groups that are at an increased risk for pica behavior are children aged 1–3 years old. Although aware that children live in Superior, ATSDR does not know whether they exhibit pica behavior. If children who exhibit pica behavior live or visit the mine site, floodplain area, or town, ATSDR suggests that parents monitor their children's behavior while playing outdoors to prevent their children from intentionally eating soil and especially avoid any soil that appears to be affected by waste tailings.

7 Community Health Concerns

Area residents expressed concerns about the Flat Creek IMM site. These concerns are discussed in the following text.

- **Comment 1:** *Can professional education to local healthcare providers and environmental health doctor be conducted? The education could include information related to the heavy metals detected in Superior and symptoms of heavy metal poisoning.*

ATSDR Response 1: Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) legislation, also known as Superfund, provides ATSDR with the authority to conduct certain public health assessment activities at hazardous waste sites. ATSDR can provide contaminant-specific training to health care providers in association with Superfund sites. In Montana, ATSDR uses a board certified Association of Occupational and Environmental Clinic (AOEC) physician, Dr. Headapohl, to deploy training materials. Health care providers and Superior residents who have questions about exposure to heavy metals can either contact Dr. Headapohl at 406-329-5746 or ATSDR staff at 1-800-CDC-INFO. When contacting ATSDR, please state you are requesting information related to the Flat Creek IMM site.

- **Comment 2:** *We are concerned that lead can be stored in the place of calcium in our bones for 10 years. Does this mean that heavy metal poisoning risks can be prolonged even after the initial exposure?*

ATSDR Response 2: Approximately 95% of lead in adults, and approximately 70% in children, resides in mineralized tissues such as bone and teeth [Barry 1975, Barry 1981]. When the body requires additional calcium, if it is not taken as a supplement or in food, the body will release stored calcium from the bones into the blood—and with it stores of accumulated lead. As stated in Section 4.4.2, internal exposure results from the constant re-equilibration of lead between the bone and the blood pool. In persons with a high body burden of lead, redistribution of lead from bone into the bloodstream can keep blood lead levels elevated for years after external exposure stops.

- **Comment 3:** *High levels of cancer, lupus, multiple sclerosis (MS), and other disease clusters are common in the Superior area.*

ATSDR Response 3: The Montana Comprehensive Cancer Control Program (MCCCP) uses data from the Montana Central Tumor Registry (MCTR), the Montana Office of Vital Statistics, and other sources to monitor trends in cancer incidence. The program publishes quarterly surveillance reports, annual tumor registry reports, and reports on special topics [MDPHHS 2009].

In addition, the state receives inquiries from public health officials and private citizens about cancer concerns. Inquiries about perceived excesses come to the MDPHHS through several channels, and some inquiries are directed to the MCTR staff. There is also a Cluster Investigation Team and protocols are in place to respond to inquiries about cancer throughout the state. In the past several years, an average of one inquiry per month has been received through all channels. The Cancer Cluster Investigation Team has been convened an average of twice a year. No excess cancer incidence was found in response to any inquiry [MDPHHS 2008].

With regard to lupus and MS, data regarding these diseases are not routinely collected by public health agencies. At this time, Montana does not have the capacity to conduct population based surveillance on chronic diseases such as lupus and MS.

- **Comment 4:** *Does “U” stands for unknown or for Uranium found in the creek besides other metals?*

ATSDR Response 4: Most environmental reports contain appropriate data qualifiers. For the Flat Creek IMM site, these reports state “U” means the analyte was not detected at or above the Contract Required Quantitation Limit (CRQL).

- ***Comment 5:** Airborne tailings are present in the dusts during the dry months in summer time. Is it safe to live by, on, or near Flat Creek? How much exposure is alright?*

ATSDR Response 5: Although waste tailings and sediment chemical-specific data are available for the Flat Creek floodplain area, air and dust data are not available. Without these data, ATSDR cannot state whether the air contains chemicals at level of public health concern.

Of note, there are a few residences outside the town of Superior and along the Flat Creek floodplain area. Because elevated levels of arsenic and lead were found in waste tailings and sediment, ATSDR recommends these residents take prudent public health measures to reduce their ingestion or inhalation of dust and soil (see Appendix D).

- ***Comment 6:** Also is dermal contact with the water and tailings in the banks of Flat Creek as well as eating fish out of Flat Creek safe? This includes the residents, campers, backpackers, hunters, fishermen, people swimming and in Flat Creek must be considered. The health risks to cemetery workers, residents, and children need to be assessed.*

ATSDR Response 6: ATSDR evaluated dermal contact with and incidental ingestion of surface water and sediment from Flat Creek. Harmful health effects are not expected for children and adults following dermal contact with and incidental ingestion of Flat Creek surface water while wading and fishing downstream of the mine site. However, ATSDR’s evaluation indicates that the potential exists for children who ingest 1 liter of Flat Creek water per day to have BLLs of health concern. In addition, ATSDR considers sediment affected by waste tailings in the floodplain area to be of public health concern if residents, particularly children, visit these areas and engage in contact-intense activities on a daily basis.

Flat Creek is considered fishable, and contains populations of rainbow trout, brook trout, and Westslope cutthroat trout [EPA 2002c]. Chemical-specific fish data are not available. Without these data, ATSDR cannot state whether the fish contain chemicals at level of public health concern. Of note, Montana’s official state website, Montana Fish, Wildlife and Parks, does not include Flat Creek on its list of fishing restrictions and closures [MFWP 2006]. Because town residents should be aware of potential contamination in Flat Creek sediment, they may take this information into account and avoid eating locally caught fish.

Within this public health assessment document, ATSDR evaluated the health risks to both children and adults. This included residents, campers, backpackers, hunters, and fishermen. Please refer to Section 5 for the chemical-specific evaluation.

- ***Comment 7:** How will Asarco’s bankruptcy affect the clean up of Flat Creek? It will cost 42 million to clean up Flat Creek for 3.5 miles on both sides of the creek to the Clark Fork River. When will Flat creek be cleaned up or contained?*

ATSDR Response 7: Monetary issues associated with cleanup efforts are outside the purview of ATSDR’s evaluation. Similarly, ATSDR is not involved in determining where and when cleanup efforts may take place.

- **Comment 8:** *What about water rights on Flat Creek? We were not told the water was contaminated and we pumped the water and drank it right from the creek for over ten years.*

ATSDR Response 8: Based on the limited data available for the creek, harmful health effects are not expected for children and adults following dermal contact and incidental ingestion of Flat Creek surface water while wading and fishing downstream of the mine site. For most chemicals, the limited data available suggest harmful health effects are not expected for resident's who use creek water for activities such as drinking, showering, bathing, cooking, and washing dishes. However, ATSDR's evaluation indicates that the potential exists for children who ingest 1 liter of Flat Creek water per day to have BLLs of health concern. In addition, average levels of antimony and lead in creek water are above regulatory guidelines. ATSDR finds that efforts to reduce drinking water exposures are considered prudent public health practice when chemical levels are above regulatory guidelines.

- **Comment 9:** *Some tailings were left in the town and how will that affect the ground water.*

ATSDR Response 9: The public drinking water supply wells are drawn from the deep groundwater aquifer, which is believed to be confined by less permeable layers of fine-grained sediments. As such, ATSDR would not expect waste tailings to affect the deep groundwater aquifer. In addition, these public supply wells are tested for water quality in accordance with federal standards.

As stated in Section 2.4, contaminated soil samples from four locations in town were run for TCLP and all four samples failed TCLP for lead. The TCLP analysis simulates landfill conditions to determine if liquid percolating through the waste could absorb and move contaminants. Although most waste tailings have been removed from town, lead may have leached into the shallow aquifer. Because several homes have wells that draw water from less than 85 feet bgs, as a protective public health measure, residents with private drinking water wells should test their wells regularly for metals and other contaminants.

- **Comment 10.** *The Flat Creek Spring can be used by the city of Superior six months out of the year. Should Superior's resident's filter their water?*

ATSDR Response 10: There is no current use of the spring as a water source; however, ATSDR is aware that the spring may be used in the future during emergencies [URS 2001]. EPA mandates the routine testing of all public drinking water supplies nationwide, which would include the spring. If desired, individuals may still use filters to improve taste and to reduce contaminants that may have leached from pipes into the water.

- **Comment 11:** *Can the lead, arsenic, and mercury levels of local kids and one Flat Creek residence be checked to see if the towns clean up helped?*

ATSDR Response 11: At this time, ATSDR would not recommend additional biological sampling, such as blood lead or urine arsenic. ATSDR feels the best method to determine whether the 2002 TCRA was successful would be to collect and analyze additional soil samples from town.

- **Comment 12:** *The Montana DEQ set the minimal risk levels at 17 ppm for arsenic and 400 ppm for lead. The EPA set the level for Helena, Montana, at 1,000 ppm for lead.*

However, the EPA set 400 ppm as the arsenic level and 3,000 ppm for lead for Superior. The people who have 2,888 ppm level of lead in their yard were told it was safe. Kids play in this dirt and the community doesn't know that these levels that the EPA set for Superior are unsafe for kids to play in. The town must be educated. Also, why are these levels set so high? There are 27 metal contaminants on Flat Creek and the dangers of all of these must be assessed.

ATSDR Response 12: Cleanup levels and action levels set by regulatory agencies vary from site to site. There are numerous factors that are considered when setting these levels such as site-specific variability in exposure, soil geochemistry, and projected land use. Regulatory agencies can also factor in other considerations such as cost, technical feasibility, compliance with state and federal regulations, and community acceptance. These factors result in large variations in proposed action levels at different sites.

ATSDR cannot directly address the factors that went into determining the action levels for Superior because we were not a part of that process. ATSDR would suggest individuals interested in further information about the action levels contact EPA directly.

As a public health advisory agency, however, ATSDR has evaluated past and current exposures to town soil. In general, ATSDR found that prior to the 2002 removal actions, arsenic and lead levels in soil were of public health concern in five residential yards, several public rights-of-way, the county fairgrounds, and the high school track. Furthermore, the potential exists that additional properties in town could still contain waste tailings material with levels of arsenic and lead of public health concern. ATSDR's finds that additional efforts, such as another mail-out to town residents or a town hall meeting, may be beneficial in determining whether additional areas of town should be characterized.

In addition, ATSDR found that the maximum arsenic levels in soil at two residences and the maximum lead levels at four residences are currently of potential health concern. ATSDR recommends additional soil sampling to determine whether the arsenic and lead levels are truly representative of these yards. As a protective measure, ATSDR suggests that if young children live at or visit these four residences, parents should monitor their children's behavior while playing outdoors to prevent their children from intentionally or inadvertently eating soil. ATSDR is working closely with EPA and local agencies to ensure these residents are made aware of ATSDR's recommendations.

8 Conclusions

Overall, ATSDR concludes that coming into frequent contact with waste tailings on the Iron Mountain Mine and Mill site, the Flat Creek floodplain, and the town of Superior could harm people's health. This is a public health hazard. ATSDR reached the following pathway-specific conclusions.

- Waste tailings deposits, and sediment affected by waste tailings, on the Iron Mountain Mine and Mill property and Flat Creek floodplain area currently show arsenic and lead levels that are of public health concern if residents, particularly children, visit these areas and engage in contact-intense⁹ activities on a daily basis.

⁹ Contact-intense activities include digging with shovels and other tools, and playing with toys (like toy trucks and action figures) on the ground surface. Adults and children can be exposed by putting soiled hands or toys in their mouth or by breathing or eating dust generated by their activities.

- Prior to removal actions, levels of arsenic and lead in soil for some town properties were of public health concern for children and adults who lived at or visited these areas and engaged in contact-intense activities on a daily basis.
- Currently, soil arsenic levels from two residential yards and soil lead levels from these two yards and two additional residential yards are of public health concern for children who live at or visit these four residences and engage in contact-intense activities on a daily basis.
- Heavy metals, including arsenic and lead, detected in soil at most residential and non-residential areas are currently not at levels of health concern for children and adults because the estimated chemical levels are below harmful effect levels. However, the potential exists for additional properties in town that were not sampled to contain waste tailings material with elevated levels of arsenic and lead and there is some uncertainty about which properties were actually remediated.
- Harmful health effects are not expected for children and adults following dermal contact with and incidental ingestion of Flat Creek surface water while wading and fishing downstream of the mine site.
- Except for lead, the limited data available suggest harmful health effects are not expected for resident's who use creek water for activities such as drinking, showering, bathing, cooking, and washing dishes. However, ATSDR's evaluation indicates that the potential exists for children who ingest 1 liter of Flat Creek water per day to have BLLs of health concern. Furthermore, average levels of antimony and lead exceed regulatory guidelines and ATSDR considers efforts to reduce drinking water exposures prudent public health practice when chemical levels are above regulatory guidelines.
- Surface water in the Hall Gulch adit area would be at levels of public health concern if the water were to be frequently ingested. However, ATSDR would not expect people to drink, or wade in, this shallow, reddish-brown, surface water.
- Chemicals detected in groundwater samples from the three town of Superior wells, Flat Creek Spring, and a private well are not at levels of public health concern. However, in the past antimony levels in Flat Creek Spring and the private well exceeded regulatory guidelines. ATSDR considers efforts to reduce drinking water exposures prudent public health practice when chemical levels are above regulatory guidelines
- ATSDR cannot evaluate groundwater exposures for residents on the north side of town who rely on private wells to obtain household water because few details are documented about these wells. With the exception of the one private well mentioned previously, no environmental data are available for these wells.

9 Recommendations

Based on ATSDR's conclusions, the agency makes the following recommendations.

1. Waste tailings-contaminated areas at the mine site and floodplain area should continue to be posted with warning signs advising the public that the soil contains arsenic and lead, which may pose a risk to public health.

2. Remedial actions should be considered to minimize exposure to waste tailings contamination, such as removal of waste tailings deposits on the mine site and floodplain area.
3. Additional characterization of town soil should be conducted to confirm the success or extent of the 2002 TCRA.
4. Additional characterization of the four residential yards should be conducted to determine whether the detected soil arsenic and lead levels are truly representative of each yard.
5. Additional efforts, such as another mail-out to town residents or a town hall meeting, should be conducted to determine which areas of town need further characterization.
6. Hall Gulch should continue to be posted with warning signs informing the public that water coming out of the adit has levels of metals that may pose a health risk.
7. Efforts to reduce drinking water exposures from Flat Creek intakes should be made because antimony and lead levels in the creek water are above regulatory guidelines.
8. Flat Creek Spring should be tested before it is used as an emergency PWS to ensure antimony levels are below EPA's MCL.
9. Private wells on the north side of town should be tested if it is determined they draw water from the shallow aquifer.
10. Residents should be made aware of prudent public health measures they can take to reduce exposures and to protect themselves, their families, and visitors (see Appendix D).

10 Public Health Action Plan

The purpose of the public health action plan is to ensure that this evaluation not only identifies potential and ongoing public health hazards, but also provides a plan of action designed to mitigate and prevent adverse human health effects that result from exposure to hazardous substances in the environment. EPA listed the Flat Creek IMM site on the NPL in April 2009, and the first phase of residential soil sampling is expected to occur in summer 2009. ATSDR staff plan to visit the site to meet with residents and the regulatory agencies during the public comment period of this document to (1) address questions the EPA, town, and community might have regarding this document and (2) maximize ATSDR's involvement in the early stages of the NPL listing.

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13 References

Aamodt RL, Rumble WF, Henkin RI. 1983. Zinc absorption in humans: Effects of age, sex, and food. In: Inglett G, ed. The nutritional bioavailability of zinc. Washington, DC: The American Chemical Society, 61-82. Cited in Agency for Toxic Substances and Disease Registry. Toxicological profile for zinc. 2005. Atlanta: US Department of Health and Human Services.

Alain G, Tousignant J, Rozenfarb E. 1993. Chronic arsenic toxicity. *Int J Dermatol* 32(12):899-901. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Angle CR, Marcus A, Cheng I-H, et al. 1984. Omaha childhood blood lead and environmental lead: A linear total exposure model. *Environ Res* 35:160-170. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for lead (update). Atlanta: US Department of Health and Human Services.

Angrisani M, Lampa E, Lisa M, et al. 1988. Vasomotor reactivity and postnatal exposure to antimony trichloride. *Curr Ther Res* 43:153-159. Cited in Agency for Toxic Substances and Disease Registry. 1992. Toxicological profile for antimony. Atlanta: US Department of Health and Human Services.

Anonymous. 1994. Antimon-III-chlorid. *Toxikologische Bewertung*. Heidelberg, Berufsgenossenschaft der chemischen Industrie 235(1994).

Apostoli P, Bartoli D, Alessio L, et al. 1999. Biological monitoring of occupational exposure to inorganic arsenic. *Occup Environ Med* 56(12):825-832. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1992a. Toxicological Profile for antimony. Atlanta: US Department of Health and Human Services. Available at: <http://www.atsdr.cdc.gov/toxprofiles/tp23.html>. Last accessed 7 January 2009.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1992b. Toxicological profile for thallium. Atlanta: US Department of Health and Human Services. Available at: <http://www.atsdr.cdc.gov/toxprofiles/tp54.html>. Last accessed 20 January 2009.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2005a. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services. Available at: <http://www.atsdr.cdc.gov/toxprofiles/tp60.html>. Last accessed 21 January 2009.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2005b. Public health assessment guidance manual (update). Atlanta: US Department of Health and Human Services.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2007a. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services. Available at: <http://www.atsdr.cdc.gov/toxprofiles/tp2.html>. Last accessed 8 January 2009.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2007b. Toxicological profile for lead (update). Atlanta: US Department of Health and Human Services. Available at: <http://www.atsdr.cdc.gov/toxprofiles/tp13.html>. Last accessed 23 January 2009.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2008a. Technical assistance report: trip report, Superior Waste Rock (aka Iron Mountain Mine and Mill), Superior, Mineral County, Montana. Atlanta: US Department of Health and Human Services. September 9, 2008.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2008b. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services. Available at: <http://www.atsdr.cdc.gov/toxprofiles/tp5.html>. Last accessed 9 January 2009.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2008c. Toxicological Profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services. Available at: <http://www.atsdr.cdc.gov/toxprofiles/tp151.html>. Last accessed 15 January 2009.

Barry PSI. 1975. A comparison of concentrations of lead in human tissue. *Br J Ind Med* 32:119-139. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for lead (update). Atlanta: US Department of Health and Human Services.

Barry PSI. 1981. Concentrations of lead in the tissues of children. *Br J Ind Med* 38:61-71. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for lead (update). Atlanta: US Department of Health and Human Services.

Barclay RK, Pencock WC, Karnofsky DA. 1953. Distribution and excretion of radioactive thallium in the chick embryo, rat, and man. *J Pharmacol Exp Ther* 107:178-187. Cited in Agency for Toxic Substances and Disease Registry. 1992. Toxicological profile for thallium. Atlanta: US Department of Health and Human Services.

Beane Freeman LE, Dennis LK, Lynch CF, et al. 2004. Toenail arsenic content and cutaneous melanoma in Iowa. *Am J Epidemiol* 160(7):679-687. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Bettley FR, O'Shea JA. 1975. The absorption of arsenic and its relation to carcinoma. *Br J Dermatol* 92:563-568. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Bickley LK, Papa CM. 1989. Chronic arsenicism with vitiligo, hyperthyroidism, and cancer. *N J Med* 86(5):377-380. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Black MR, Medeiros DM, Brunett E, et al. 1988. Zinc supplements and serum lipids in young adult white males. *Am J Clin Nutr* 47:970-975. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Borgoño JM, Greiber R. 1972. Epidemiological study of arsenicism in the city of Antofagasta. *Trace Subst Environ Health* 5:13-24. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Bothwell TH, Charlton RW, Cook JD, Finch CA. 1979. Iron metabolism in man. St. Louis: Oxford: Blackwell Scientific. Cited in National Institutes of Health, Office of Dietary Supplements. Dietary supplement fact sheet: iron. Available at: <http://dietary-supplements.info.nih.gov/factsheets/iron.asp#en1>. Last accessed 13 March 2008.

Bowen HJM. 1966. Trace elements in biochemistry. New York, London, Academic Press. Cited in International Programme on Chemical Safety. 1996. Thallium. Environmental health criteria. Vol. 182. World Health Organization, Geneva, Switzerland.

Bowen HJM. 1979. Environmental chemistry of the elements. New York, London, Academic Press. Cited in International Programme on Chemical Safety. 1996. Thallium. Environmental health criteria. Vol. 182. World Health Organization, Geneva, Switzerland.

Brock C, Curry H, Hanna C, Knipfer M, Taylor L. 1985. Adverse effects of iron supplementation: A comparative trial of wax-matrix iron preparation and conventional ferrous sulfate tablets. *Clin Ther* 7:568-573. Cited in National Academy of Sciences. 2001. Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. Washington, DC: National Academy Press.

Brumsack H-J. 1977. Potential metal pollution in grass and soil samples around brickworks. *Environ Geol*, 2: 33-41. Cited in International Programme on Chemical Safety. 1996. Thallium. Environmental health criteria. Vol. 182. World Health Organization, Geneva, Switzerland.

Brzóška MM, Moniuszko-Jakoniuk J. 2005a. Disorders in bone metabolism of female rats chronically exposed to cadmium. *Toxicol Appl Pharmacol* 202(1):68-83. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

Brzóška MM, Moniuszko-Jakoniuk J. 2005b. Bone metabolism of male rats chronically exposed to cadmium. *Toxicol Appl Pharmacol* 207(3):195-211. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

Brzóška MM, Moniuszko-Jakoniuk J. 2005c. Effect of low-level lifetime exposure to cadmium on calciotropic hormones in aged female rats. *Arch Toxicol* 79(11):636-646. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

Buchet JP, Lauwerys R, Roels H. 1981. Comparison of the urinary excretion of arsenic metabolites after a single oral dose of sodium arsenite, monomethylarsonate or dimethylarsinate in man. *Int Arch Occup Environ Health* 48:71-79. Cited in Agency for Toxic Substances and

Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Bureau of the Census. 2001. US Census Bureau web site. Population estimates, cities and towns. Available at: <http://www.census.gov/>. Last accessed 15 September 2006.

Casteel SW, Brown LD, Dunsmore ME, Weis CP, Henningsen GM, Hoffman E, Brattin WJ, Hammon TL. 1997. Relative bioavailability of arsenic in mining wastes. Document control number: 4500-88-AORH. Prepared for US Environmental Protection Agency, Region VIII, Denver, Colorado. Cited in Battelle and Exponent. 2000. Final guide for incorporating bioavailability adjustments into human health and ecological risk assessments at US Navy and Marine Corps Facilities. Part 1: overview of metals bioavailability. Prepared for Naval Facilities Engineering Service Center and Engineering Field Activity West.

[CDC] Centers for Disease Control and Prevention. 2005. Preventing lead poisoning in young children: a statement by the Centers for Disease Control and Prevention. Atlanta: US Department of Health and Human Services.

Cebrián ME, Albores A, Aguilar M, et al. 1983. Chronic arsenic poisoning in the north of Mexico. *Hum Toxicol* 2:121-133. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Chattopadhyay A, Jervis RE. 1974. Multielement determination in market-garden soils by instrumental photon activation analysis. *Anal Chem*, 46: 1630-1639. Cited in International Programme on Chemical Safety. 1996. Thallium. Environmental health criteria. Vol. 182. World Health Organization, Geneva, Switzerland.

Chen YC, Guo YL, Su HJ, et al. 2003. Arsenic methylation and skin cancer risk in southwestern Taiwan. *J Occup Environ Med* 45(3):241-248. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Cooper WC. 1984. The health implications of increased manganese in the environment resulting from the combustion of fuel additives: A review of the literature. *J Toxicol Environ Health* 14:23-46. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

Coplin M, Schuette S, Leichtmann G, Lashner B. 1991. Tolerability of iron: A comparison of bis-glycino iron II and ferrous sulfate. *Clin Ther* 13:606-612. Cited in National Academy of Sciences. 2001. Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. Washington, DC: National Academy Press.

Cousins RJ. 1996. Zinc. In: Filer LJ, Ziegler EE, eds. *Present Knowledge in Nutrition*, 7th ed. Washington, DC: International Life Science Institute-Nutrition Foundation. Pp. 293-306. Cited in National Academy of Sciences. 2001. Cited in National Academy of Sciences. 2001. Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron,

manganese, molybdenum, nickel, silicon, vanadium, and zinc. Washington, DC: National Academy Press.

Creclius EA. 1977. Changes in the chemical speciation of arsenic following ingestion by man. *Environ Health Perspect* 19:147-150. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Davidsson L. 2003. Approaches to improve iron bioavailability from complementary foods. *J Nutr* 133:1560S-2S. Cited in National Institutes of Health, Office of Dietary Supplements. Dietary supplement fact sheet: iron. Available at: <http://dietary-supplements.info.nih.gov/factsheets/iron.asp#en1>. Last accessed 13 March 2008.

Davidsson L, Cederblad A, Hagebo E, et al. 1988. Intrinsic and extrinsic labeling for studies of manganese absorption in humans. *J Nutr* 118:1517-1524. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

Davies NT. 1980. Studies on the absorption of zinc by rat intestine. *Br J Nutr* 43:189-203. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Davis A, Ruby MV, Bergstrom PD. 1992. Bioavailability of arsenic and lead in soils from the Butte, Montana, mining district. *Environ Sci Technol*. 26(3):461-468. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Davis CD, Milne DB, Nielsen FH. 2000. Changes in dietary zinc and copper affect zinc-status indicators of postmenopausal women, notably, extracellular superoxide dismutase and amyloid precursor proteins. *Am J Clin Nutr* 71:781-788. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

[DHEW] US Department of Health, Education, and Welfare, Bureau of Water Hygiene. 1970. Community water supply study. Analysis of national survey findings. Cincinnati, OH: NTIS No. PB-214982. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

Dieter M. 1992. NTP technical report on the toxicity studies of Toxicity Studies of Antimony Potassium Tartrate (CAS No. 28300-74-5) in F344/N Rats And B6C3F1 Mice (Drinking Water and Intraperitoneal Injection Studies). Toxic Rep Ser #: 1-D2.

Downs WL, Scott JK, Steadman LT, et al. 1960. Acute and sub-acute toxicity studies of thallium compounds. *Am Ind Hyg Assoc J* 21:399-406. Cited in Agency for Toxic Substances and Disease Registry. 1992. Toxicological profile for thallium. Atlanta: US Department of Health and Human Services.

Dunn, JT. 1928. A curious case of antimony poisoning. *Analyst*. 53: 532-533. Cited in US Environmental Protection Agency. 2008. Integrated Risk Information System (IRIS). Summary for antimony. Available at: <http://www.epa.gov/ncea/iris/subst/0006.htm>. Last accessed 7 April 2008.

Eckel WP, Jacob TA. 1988. Ambient levels of 24 dissolved metals in US surface and grounds waters. Presented before the Division of Environmental Chemistry, American Chemical Society, Los Angeles, CA, September 25-30, 1988. Preprint of extended abstract. Alexandria, VA: Viar and Company, 371-372. Cited in Agency for Toxic Substances and Disease Registry. 1992. Toxicological profile for antimony. Atlanta: US Department of Health and Human Services. Also cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for lead (update). Atlanta: US Department of Health and Human Services.

Eckel WP, Langley WD. 1988. A background-based ranking technique for assessment of elemental enrichment in soils at hazardous waste sites. In: Superfund '88: Proceedings of the 9th National Conference. Washington, DC, 282-286. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

Eisenstein RS, Blemings KP. 1998. Iron regulatory proteins, iron responsive elements and iron homeostasis. *J Nutr* 128(12):2295-8.

Eisler R. 1985. Cadmium hazards to fish, wildlife, and invertebrates: A synoptic view. *US Fish Wild Serv Biol Rep* 85(1.2) 1-46. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

Elinder CG. 1985. Cadmium: Uses, occurrence and intake. In: Friberg L, Elinder CG, Kjellstrom T, et al., eds. *Cadmium and health: A toxicological and epidemiological appraisal*. Vol. I. Exposure, dose, and metabolism. Effects and response. Boca Raton, FL: CRC Press, 23-64. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

Elinder CG. 1992. Cadmium as an environmental hazard. *IARC Sci Pub* 1118:123-132. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

[EPA] US Environmental Protection Agency. 1980a. Ambient water quality criteria for thallium. Washington, DC: Office of Water Regulations and Standards. EPA-440/5-80-074. NTIS No. PB81-117848. Cited in Agency for Toxic Substances and Disease Registry. 1992. Toxicological profile for thallium. Atlanta: US Department of Health and Human Services.

[EPA] U.S. Environmental Protection Agency. 1980b. Exposure and risk assessment for zinc. Washington, DC: US Environmental Protection Agency, Office of Water Regulations and Standards (WH-553). EPA440481016. PB85212009. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

[EPA] US Environmental Protection Agency. 1981. Community health associated with arsenic in drinking water in Millard County, Utah. Cincinnati, OH: US Environmental Protection Agency, Health Effects Research Laboratory. EPA600/181064. PB82108374. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

[EPA] US Environmental Protection Agency. 1982a. Exposure and risk assessment for arsenic. Washington, DC: US Environmental Protection Agency, Office of Water Regulations and Standards. PB85221711. EPA440485005. 1.14.68. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

[EPA] US Environmental Protection Agency. 1982b. Inductively coupled plasma-atomic emission spectrometric method for trace element analysis of water and wastes - method 200.7. Cincinnati, OH: US Environmental Protection Agency, Environmental Monitoring and Support Laboratory. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

[EPA] US Environmental Protection Agency. 1984. Health assessment document for manganese. Final draft. Cincinnati, OH: US Environmental Protection Agency, Office of Research and Development. EPA-600/8-83-013F. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

[EPA] US Environmental Protection Agency. 1985a. Cadmium contamination of the environment: An assessment of nationwide risk. Washington, DC: US Environmental Protection Agency, Office of Water Regulations and Standards. EPA-440/4-85-023. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

[EPA] US Environmental Protection Agency. 1985b. Locating and emitting air emissions from sources of manganese. Research Triangle Park, NC: US Environmental Protection Agency, Office of Air Quality Planning and Standards. EPA-450/4-84-007h. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

[EPA] US Environmental Protection Agency. 1986a. Subchronic (90-day) toxicity of thallium sulfate in Sprague-Dawley rats. Office of Solid Waste, Washington, DC. Cited in US Environmental Protection Agency. Integrated Risk Information System (IRIS). Thallium acetate (CASRN 563-68-8), thallium carbonate (CASRN 6533-73-9), thallium chloride (CASRN 7791-12-0), thallium nitrate (CASRN 10102-45-1), thallium (I) sulfate (CASRN 7446-18-6). Available at: <http://www.epa.gov/ncea/iris/subst/0111.htm>, <http://www.epa.gov/ncea/iris/subst/0112.htm>, <http://www.epa.gov/ncea/iris/subst/0113.htm>, <http://www.epa.gov/ncea/iris/subst/0114.htm>, <http://www.epa.gov/ncea/iris/subst/0116.htm>. Last accessed 17 March 2008.

[EPA] US Environmental Protection Agency. 1986b. Air quality criteria for lead. Research Triangle Park, NC: US Environmental Protection Agency, Office of Research and Development,

Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office. EPA600883028F. Cited in Agency for Toxic Substances and Disease Registry. 2007b. Toxicological profile for lead (update). Atlanta: US Department of Health and Human Services.

[EPA] US Environmental Protection Agency, Office of Solid Waste and Emergency Response. 1988. Health and environmental effects document for thallium and compounds. Cincinnati, OH: ECAO-CIN-G031. Cited in Agency for Toxic Substances and Disease Registry. 1992. Toxicological profile for thallium. Atlanta: US Department of Health and Human Services.

[EPA] US Environmental Protection Agency. 1991. Maximum contaminant level goals and national primary drinking water regulations for lead and copper. Fed Regist 56:26461-26564. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for lead (update). Atlanta: US Department of Health and Human Services.

[EPA] US Environmental Protection Agency. 1994. Guidance manual for the integrated exposure uptake biokinetic model for lead in children. NTIS #PB93-963510, EPA 9285.7-15-1. Office of Solid Waste and Emergency Response. Washington, DC.

[EPA] US Environmental Protection Agency. 1998. Clarification to the 1994 revised interim soil lead (Pb) guidance for CERCLA sites and RCRA corrective action facilities. OSWER Directive No. 9200.4-27P, Document no. EPA/540/F-98/030, PB98-963244. Washington, DC: Office of Solid Waste and Emergency Response.

[EPA] US Environmental Protection Agency. 2001a. Electronic mail from Rosemary Rowe, EPA Montana Office, summarizing past and current activities related to the Iron Mountain Mill site and town of Superior, Mineral County, Montana. August 7, 2001.

[EPA] US Environmental Protection Agency. 2001b. Electronic mail from Jay Sinnott, EPA Montana Office, with drinking water supply information for the town of Superior, Mineral County, Montana. August 9, 2001.

[EPA] US Environmental Protection Agency. 2002a. Analytical results report for focused site inspection. Iron Mountain Mill, Superior, Mineral County, Montana. January 24, 2002.

[EPA] US Environmental Protection Agency. 2002b. Electronic mail from Tien Nguyen, On-Scene Coordinator, ERT, regarding the letter to be sent to Superior residents. February 28, 2002.

[EPA] US Environmental Protection Agency. 2002c. Action memorandum, documentation of a time-critical removal action at Superior Waste Rock site near and in the town of Superior in Mineral County, Montana. From Tien Nguyen, On-Scene Coordinator, ERT. August 2, 2002.

[EPA] US Environmental Protection Agency. 2002d. Sampling and analysis report. Superior Waste Rock, Superior, Mineral County, Montana. August 16, 2002.

[EPA] US Environmental Protection Agency. 2002e. Initial pollution report, Superior Waste Rock, Superior, Mineral County, Montana. August 27, 2002.

[EPA] US Environmental Protection Agency. 2002f. Memorandum, Superior Waste Rock confirmation samples, Superior, Mineral County, Montana. From John A. McKeown, On-Scene Coordinator. October 1, 2002.

[EPA] US Environmental Protection Agency. 2003a. Final pollution report, Superior Waste Rock, Superior, Mineral County, Montana. June 23, 2003.

[EPA] US Environmental Protection Agency. 2003b. Assessing intermittent or variable exposures at lead sites. OSWER Directive No. 9285.7-76, Document no. EPA-540-R-03-008. Washington, DC: Office of Solid Waste and Emergency Response.

[EPA] US Environmental Protection Agency. 2003c. Superfund lead-contaminated residential sites handbook. OSWER Directive No. 9285.7-50. Washington, DC: Office of Solid Waste and Emergency Response.

[EPA] US Environmental Protection Agency. 2005. STORET data warehouse. US Environmental Protection Agency. Available at: http://www.epa.gov/storet/dw_home.html. April 8, 2005. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

[EPA] US Environmental Protection Agency. 2006. Provisional peer reviewed toxicity values for iron and compounds (CASRN 7439-89-6). Derivation of subchronic and chronic oral RfDs. September 11, 2006.

[EPA] US Environmental Protection Agency. 2008. Integrated Risk Information System (IRIS). Files accessed: antimony, arsenic, cadmium, manganese, and zinc. Available at: <http://www.epa.gov/ncea/iris>. Last accessed 23 January 2009.

[FDA] US Food and Drug Administration. 1997. Preventing iron poisoning in children. FDA backgrounder. January 15, 1997. Available at: <http://www.cfsan.fda.gov/~dms/bgiron.html>. Last accessed 13 March 2008.

Fellicetti SW, Thomas RG, McClellan RO. 1974. Metabolism of two valence states of inhaled antimony in hamsters. *Am Ind Hyg Assoc J* 35:292-300. Cited in Agency for Toxic Substances and Disease Registry. 1992. Toxicological profile for antimony. Atlanta: US Department of Health and Human Services.

Fischer PWF, Giroux A, L'Abbe AR. 1984. Effect of zinc supplementation on copper status in adult man. *Am J Clin Nutr* 40:743-746. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Flynn HC, Meharg AA, et al. 2003. Antimony bioavailability in mine soils. *Environ Pollut* 124(1): 93-100.

Franzblau A, Lilis R. 1989. Acute arsenic intoxication from environmental arsenic exposure. *Arch Environ Health* 44(6):385-390. Cited in Agency for Toxic Substances and Disease

Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Freeland-Graves JH, Friedman BJ, Han W, et al. 1982. Effect of zinc supplementation on plasma highdensity lipoprotein and zinc. *Am J Clin Nutr* 35:988-992. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Freeland-Graves JH, Bales CW, Behmardi F. 1987. Manganese requirements of humans. Nutritional bioavailability of manganese. American Chemical Society, 90-104. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

Freeman GB, Johnson JD, Killinger JM, Liao SC, Feder PI, Davis AO, Ruby MV, Chaney RL, Lovre SC, Bergstrom PD. 1993. Bioavailability of arsenic in soil impacted by smelter activities following oral administration in rabbits. *Fundam Appl Toxicol*. 21(1):83-88. Cited in Battelle and Exponent. 2000. Final guide for incorporating bioavailability adjustments into human health and ecological risk assessments at US Navy and Marine Corps Facilities. Part 1: overview of metals bioavailability. Prepared for Naval Facilities Engineering Service Center and Engineering Field Activity West.

Freeman GB, Schoof RA, Ruby MV, Davis AO, Dill JA, Liao SC, Lapin CA, Bergstrom PD. 1995. Bioavailability of arsenic in soil and house dust impacted by smelter activities following oral administration in cynomolgus monkeys. *Fundam Appl Toxicol*. 28(2):215-222. Cited in Battelle and Exponent. 2000. Final guide for incorporating bioavailability adjustments into human health and ecological risk assessments at US Navy and Marine Corps Facilities. Part 1: overview of metals bioavailability. Prepared for Naval Facilities Engineering Service Center and Engineering Field Activity West.

Frykman E, Bystrom M, Jansson U, Edberg A, Hansen T. 1994. Side effects of iron supplements in blood donors: Superior tolerance of heme iron. *J Lab Clin Med* 123:561-564. Cited in National Academy of Sciences. 2001. Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. Washington, DC: National Academy Press.

Gale NL, Adams CD, Wixson BG, et al. 2002. Lead concentrations in fish and river sediments in the old lead belt of Missouri. *Environ Sci Technol* 36:4262-4268. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for lead (update). Atlanta: US Department of Health and Human Services.

Gale NL, Adams CD, Wixson BG, et al. 2004. Lead, zinc, copper, and cadmium in fish and sediments from the Big River and Flat River Creek of Missouri's Old Lead Belt. *Environ Geochem Health* 26:3749. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for lead (update). Atlanta: US Department of Health and Human Services.

Gerber GB, Maes J, Eykens B. 1982. Transfer of antimony and arsenic to the developing organism. *Arch Toxicol* 49:159-168. Cited in Agency for Toxic Substances and Disease

Registry. 1992. Toxicological profile for antimony. Atlanta: US Department of Health and Human Services.

Graedel TE. 1978. Inorganic elements, hydrides, oxides, and carbonates. In: Chemical compounds in the atmosphere. New York, NY: Academic Press, 35-41, 44-49. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

Groen, K., H. Vaessen, J.J.G. Kliest, J.L.M. deBoer, T.V. Ooik, A. Timmerman, and F.F. Vlug. 1994. Bioavailability of Inorganic Arsenic from Bog Ore-Containing Soil in the Dog. *Environ. Health Perspect.*, 102(2): 182-184. Cited in Battelle and Exponent. 2000. Final guide for incorporating bioavailability adjustments into human health and ecological risk assessments at US Navy and Marine Corps Facilities. Part 1: overview of metals bioavailability. Prepared for Naval Facilities Engineering Service Center and Engineering Field Activity West.

Guha Mazumder DN, Chakraborty AK, Ghose A, et al. 1988. Chronic arsenic toxicity from drinking tubewell water in rural west Bengal. *Bull WHO* 66(4):499-506. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Haines RC. 1984. Environmental contamination-surveys of heavy metals in urban soils and hazard assessment. *Trace Substances in Environmental Health* 18:450-460. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Hallberg L, Hulthen L, Gramatkovski E. 1997. Iron absorption from the whole diet in men: how effective is the regulation of iron absorption? *Am J Clin Nutr* 66:347-56. Cited in National Institutes of Health, Office of Dietary Supplements. Dietary supplement fact sheet: iron. Available at: <http://dietary-supplements.info.nih.gov/factsheets/iron.asp#en1>. Last accessed 13 March 2008.

Haque R, Mazumder DN, Samanta S, et al. 2003. Arsenic in drinking water and skin lesions: Dose-response data from West Bengal, India. *Epidemiology* 14(2):174-182. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Harrington JM, Middaugh JP, Morse DL, et al. 1978. A survey of a population exposed to high concentrations of arsenic in well water in Fairbanks, Alaska. *Am J Epidemiol* 108(5):377-385. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Haupt TA, Wiersma JH, Goldring JM. 1996. Health effects of ingesting arsenic-contaminated groundwater. *Wis Med J* 95(2):100-104. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Health Canada. 1993. Canadian Environmental Protection Act. Priority substances list assessment report: arsenic and its compounds. Government of Canada, Environment Canada.

Available at: http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/psl1-lsp1/arsenic_comp/index_e.html. Last accessed 13 March 2008.

Hsueh YM, Cheng GS, Wu MM, et al. 1995. Multiple risk factors associated with arsenic-induced skin cancer: Effects of chronic liver disease and malnutritional status. *Br J Cancer* 71(1):109-14. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Hunt JR, Lykken GI, Mullen Lk. 1991. Moderate and high amounts of protein from casein enhance human absorption of zinc from whole-wheat or white rolls. *Nutr Res* 11(5):413-418. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

[IARC] International Agency for Research on Cancer. 1980. Arsenic and arsenic compounds. In: IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Some metals and metallic compounds. IARC monographs, Vol. 23. Lyon, France: World Health Organization. International Agency for Research on Cancer, 39-141. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

[IARC] International Agency for Research on Cancer. 1993. Cadmium and certain cadmium compounds. In: IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Beryllium, cadmium, mercury and exposures in the glass manufacturing industry. IARC monographs, Vol. 58. Lyon, France: World Health Organization. International Agency for Research on Cancer, 119-146, 210-236. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

[IPCS] International Programme on Chemical Safety. 1996. Thallium. Environmental health criteria. Vol. 182. World Health Organization, Geneva, Switzerland. Available at: <http://www.inchem.org/documents/ehc/ehc/ehc182.htm>. Last accessed 17 March 2008.

Istfan NW, Janghorbani M, Young VR. 1983. Absorption of stable ^{70}Zn in healthy young men in relation to zinc intake. *Am J Clin Nutr* 38:187-194. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Johnson PE, Hunt JR, Ralston NV. 1988. The effect of past and current dietary Zn intake on Zn absorption and endogenous excretion in the rat. *J Nutr* 118:1205-1209. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Kallman, DA, et al. 1990. The effect of variable environmental arsenic contamination on urinary concentrations of arsenic species. *Environ Health Perspect*. 89:145-51.

Kjellstrom T, Borg K, Lind B. 1978. Cadmium in feces as an estimator of daily cadmium intake in Sweden. *Environ Res* 15:242-251. Cited in Agency for Toxic Substances and Disease

Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

Konz J, Walker P. 1979. An assessment of cadmium in drinking water from a multi-media perspective. Report to US Environmental Protection Agency by The Mitre Corporation, McLean, VA. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

Kopp JF, Kroner RC. 1967. Trace metals in waters of the United States. A five year summary of trace metals in rivers and lakes of the United States (Oct. 1, 1962 - Sept. 30, 1967). Cincinnati, OH: US Department of the Interior, Federal Water Pollution Control Administration. NTIS No. PB-215680. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

Kosnet MJ. 2001. Lead. In: Ford MD, Delaney KA, Ling LJ, Erickson T, editors. *Clinical Toxicology*. Philadelphia, Pennsylvania: W.B. Saunders Company. p. 728.

Kotsonis FN, Klaassen CD. 1978. The relationship of metallothionein to the toxicity of cadmium after prolonged administration to rats. *Toxicol Appl Pharmacol* 46:39-54. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

Kurtzweil P. 1993. Daily values encourage health diet. US Food and Drug Administration. May 1993. Available at: <http://www.fda.gov/fdac/special/foodlabel/dvs.html>. Last accessed 13 March 2008.

Lacey RF, Moore MR, Richards WN. 1985. Lead in water, infant diet and blood: The Glasgow duplicate diet stud. *Sci Total Environ* 41:235-257. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for lead (update). Atlanta: US Department of Health and Human Services.

[LANL] Los Alamos National Laboratory, Chemistry Division. 2003. Periodic table of the elements: iron. Available at: <http://periodic.lanl.gov/elements/26.html>. Last updated on 15 December 2003. Last accessed 13 March 2008.

Lewis DR, Southwick JW, Ouellet-Hellstrom R, et al. 1999. Drinking water in Utah: A cohort mortality study. *Environ Health Perspect* 107(5):359-365. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Liguori L. 1993. Iron protein succinylate in the treatment of iron deficiency: Controlled, double-blind, multicenter clinical trial on over 1,000 patients. *Int J Clin Pharmacol Ther Toxicol* 31:103-123. Cited in National Academy of Sciences. 2001. *Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc*. Washington, DC: National Academy Press.

Lokken P, Birkeland JM. 1979. Dental discolorations and side effects with iron and placebo tablets. *Scand J Dent Res* 87:275–278. Cited in National Academy of Sciences. 2001. Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. Washington, DC: National Academy Press.

Looker A, CT Sempos, C Johnson, and EA Yetley. 1988. Vitamin-mineral supplement use: Association with dietary intake and iron status of adults. *J. Am. Diet. Assoc.* 88:808–814. Cited in US Environmental Protection Agency. Provisional peer reviewed toxicity values for iron and compounds (CASRN 7439-89-6). Derivation of subchronic and chronic oral RfDs. September 11, 2006.

Lüchtrath H. 1983. The consequences of chronic arsenic poisoning among Moselle wine growers: Pathoanatomical investigations of post-mortem examinations performed between 1960 and 1977. *J Cancer Res Clin Oncol* 105:173-182. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Marafante E, Vahter M, Norin H, et al. 1987. Biotransformation of dimethylarsinic acid in mouse, hamster and man. *J Appl Toxicol* 7(2):111-117. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Marcus WL, Rispin AS. 1988. Threshold carcinogenicity using arsenic as an example. In: Cothorn CR, Mehlman MA, Marcus WL, eds. *Advances in modern environmental toxicology*. Vol. XV: Risk assessment and risk management of industrial and environmental chemicals. Princeton, NJ: Princeton Scientific Publishing Co., 133-158. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Marmo E, Matera MG, Acampora R, et al. 1987. Prenatal and postnatal metal exposure: Effect on vasomotor reactivity development of pups. *Curr Ther Res* 42:823-838. Cited in Agency for Toxic Substances and Disease Registry. 1992. Toxicological profile for antimony. Atlanta: US Department of Health and Human Services.

[MCEHP] Mineral County Environmental Health and Planning. 2002a. Environmental health notice, Flat Creek arsenic and lead exposure prevention. Superior, MT. February 8, 2002.

[MCEHP] Mineral County Environmental Health and Planning. 2002b. Environmental health notice, Flat Creek arsenic and lead exposure prevention. Superior, MT. February 23, 2002.

[MCEHP] Mineral County Environmental Health and Planning. 2002c. March 12th letter from Wayne Marchwick, MCEHP, to the Town of Superior, Town Council Members, regarding contaminated tailings. Superior, MT. March 12, 2002.

[MCEHP] Mineral County Environmental Health and Planning. 2002d. March 25rd facsimile transmittal from Wayne Marchwick, MCEHP, regarding the environmental health notice. Superior, MT. March 23, 2002.

McLellan JS, Flanagan PR, Chamberlain MJ, et al. 1978. Measurement of dietary cadmium absorption in humans. *J Toxicol Environ Health* 4:131-138. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

[MDEQ] Montana Department of Environmental Quality. 2000. November 6th letter from Judy Reese, MDEQ, regarding the Iron Mountain Mine, Superior, Montana, Water Quality Sampling results. Helena, MT. November 6, 2000.

[MDEQ] Montana Department of Environmental Quality. 2003. Source water delineation and assessment report, town of Superior, public water supply. July 31, 2003.

[MDEQ] Montana Department of Environmental Quality. 2004a. Historical narrative: Iron Mountain Mine. Available at: <http://www.deq.state.mt.us/AbandonedMines/linkdocs/techdocs/137tech.asp>. Last accessed 21 July 2004.

[MDEQ] Montana Department of Environmental Quality. 2004b. Iron Mountain Mill Facility CERCA listing responsiveness summary. October 4, 2004.

[MDEQ] Montana Department of Environmental Quality. 2005. Action level for arsenic in surface soil. Montana Department of Environmental Quality, Remediation Division. Available at: <http://www.deq.mt.gov/StateSuperfund/PDFs/ArsenicPositionPaper.pdf>. Last assessed 8 January 2009.

[MDEQ] Montana Department of Environmental Quality. 2007. Sampling Data, Superior Public Water Supply System. Available at: <http://deq.mt.gov/wqinfo/pws/reports.asp>. Data retrieved 8 August 2007.

[MDPHHS] Montana Department of Public Health and Human Services. 2008. Quarterly surveillance report. July, 2008. Environmental carcinogens and sentinel event monitoring. Available at: http://www.dphhs.mt.gov/PHSD/cancer-control/documents/July2008Environmentalcarcinogensandsentineleventmonitoring_000.pdf. Last accessed 27 January 2009.

[MDPHHS] Montana Department of Public Health and Human Services. 2009. Cancer data & statistics. Available at: <http://www.dphhs.mt.gov/PHSD/cancer-control/cancer-control-data-stats.shtml>. Last accessed 27 January 2009.

Mena I, Horiuchi K, Burke K, et al. 1969. Chronic manganese poisoning: Individual susceptibility and absorption of iron. *Neurology* 19:1000-1006. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

[MFG] MFG Inc. 2000. Department of Environmental Quality Remediation Division. Water Quality Data – Flat Creek Area. October 6, 2000.

[MFWP] Montana Fish, Wildlife and Parks. 2006. Montana's official state website, general fishing seasons and hours. Available at: <http://fwp.mt.gov/fishing/regulations/seasons.html>. Last accessed 15 September 2006.

Milne DB, Davis CD, Nielsen FH. 2001. Low dietary zinc alters indices of copper function and status in postmenopausal women. *Nutrition* 17:701-708. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Miret S, Simpson RJ, McKie AT. 2003. Physiology and molecular biology of dietary iron absorption. *Annu Rev Nutr* 23:283-301. Cited in National Institutes of Health, Office of Dietary Supplements. Dietary supplement fact sheet: iron. Available at: <http://dietary-supplements.info.nih.gov/factsheets/iron.asp#en1>. Last accessed 13 March 2008.

Mitra SR, Mazumder DN, Basu A, et al. 2004. Nutritional factors and susceptibility to arsenic-caused skin lesions in West Bengal, India. *Environ Health Perspect* 112(10):1104-1109. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Mizuta N, Mizuta M, Ito F, et al. 1956. An outbreak of acute arsenic poisoning caused by arsenic-contaminated soy-sauce (shōyu): A clinical report of 220 cases. *Bull Yamaguchi Med Sch* 4(2-3):131-149. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Monier-Williams GW. 1934. Antimony in enamelled hollow-ware. Report on Public Health and Medical Subjects, No. 73, Ministry of Health, London. p. 18. Cited in US Environmental Protection Agency. 2008. Integrated Risk Information System (IRIS). Summary for antimony. Available at: <http://www.epa.gov/ncea/iris/subst/0006.htm>. Last accessed 7 April 2008.

Monson ER. 1988. Iron and absorption: dietary factors which impact iron bioavailability. *J Am Dietet Assoc.* 88:786-90. Cited in National Institutes of Health, Office of Dietary Supplements. Dietary supplement fact sheet: iron. Available at: <http://dietary-supplements.info.nih.gov/factsheets/iron.asp#en1>. Last accessed 13 March 2008.

Morris JS, Schmid M, Newman S, et al. 1974. Arsenic and noncirrhotic portal hypertension. *Gastroenterology* 66(1):86-94. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

[NAS] National Academy of Sciences. 1973. Medical and biological effects of environmental pollutants: manganese. Washington, DC: National Academy of Sciences. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

[NAS] National Academy of Sciences. 1977. Drinking water and health--inorganic solutes. National Academy of Sciences. Washington, DC: National Academy Press, 1:205-229, 299-304, 315-316, 447-460. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

[NAS] National Academy of Sciences. 1980. Drinking water and health. Vol. 3. Washington, DC: National Academy Press, 331-337. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

[NAS] National Academy of Sciences. 2001. Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. Washington, DC: National Academy Press. Available at: <http://books.nap.edu/books/0309072794/html/index.html>. Last accessed 14 March 2008.

[NCDC] National Climatic Data Center. 2006. Climatology of the United States No. 20, 1971-2000. National Oceanic and Atmospheric Administration. Available at: <http://cdo.ncdc.noaa.gov/climatenormals/clim20/mt/248043.pdf>. Last accessed September 14, 2006.

[NHMRC] National Health and Medical Research Council. 2009. Nutrient reference values for Australia and New Zealand. Manganese. Available at: <http://www.nrv.gov.au/nutrients/manganese.htm>. Last accessed 5 June 2009.

[NRC] National Research Council. 1989. Recommended dietary allowances. 10th edition. Food and Nutrition Board, National Research Council. Washington, DC: National Academy Press. p. 230-235. Cited in US Environmental Protection Agency. Integrated Risk Information System (IRIS). Manganese (CASRN 7439-96-5). Available at: <http://www.epa.gov/ncea/iris/subst/0373.htm>. Last accessed 14 March 2008.

[NRC] National Research Council. 1999. Committee on Arsenic in Drinking Water. Arsenic in drinking water. Washington, DC: National Academy Press.

[NRC] National Research Council. 2001. Arsenic in drinking water. 2001 Update. Washington, DC: National Academy Press. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Page GW. 1981. Comparison of groundwater and surface water for patterns and levels of contamination by toxic substances. Environ Sci Technol 15(12):1475-1481. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Pennington JA, Young BE, Wilson DB. 1989. Nutritional elements in U.S. diets: results from the Total Diet Study, 1982 to 1986. J Am Diet Assoc 89:659-664. Cited in US Environmental Protection Agency. 2008. Integrated Risk Information System (IRIS). Zinc and compounds (CASRN 7440-66-6). Available at: <http://www.epa.gov/ncea/iris/subst/0426.htm>. Last accessed 17 March 2008.

Poon RI, Chu, et al. 1998. Effects of antimony on rats following 90-day exposure via drinking water. Food Chem Toxicol 36(1): 21-35.

Prasad AS, Brewer GJ, Schoomaker EB, et al. 1978. Hypocupremia induced by zinc therapy in adults. *JAMA* 240:2166-2168. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Rahola T, Aaran R-K, Miettinen JK. 1973. Retention and elimination of ^{115}mCd in man. In: Health physics problems of internal contamination. Budapest: Akademia 213-218. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

Reinhold JG, Faradji B, Abadi P, et al. 1991. Decreased absorption of calcium, magnesium, and phosphorous by humans due to increased fiber and phosphorous consumption as wheat bread. *Nutr Rev* 49(7):204-206. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Robertson FN. 1989. Arsenic in ground-water under oxidizing conditions, southwest United States. *Environ Geochem Health* 11:171-185. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Rodriguez, R.R., N.T. Basta, S.W. Casteel, and L.W. Pace. 1999. An In Vitro Gastrointestinal Method to Estimate Bioavailable Arsenic in Contaminated Soils and Solid Media. *Environ. Sci. Technol.*, 33(4): 642-649. Cited in Battelle and Exponent. 2000. Final guide for incorporating bioavailability adjustments into human health and ecological risk assessments at US Navy and Marine Corps Facilities. Part 1: overview of metals bioavailability. Prepared for Naval Facilities Engineering Service Center and Engineering Field Activity West.

Rope SK, Arthur WJ, Craig TH, et al. 1988. Nutrient and trace elements in soil and desert vegetation of southern Idaho. *Environmental Monitoring and Assessment* 10:1-24. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

Rossi F, Acampora R, Vacca C, et al. 1987. Prenatal and postnatal antimony exposure in rats: Effect on vasomotor reactivity development of pups. *Teratogen Carcinogen Mutagen* 7:491-496. Cited in Agency for Toxic Substances and Disease Registry. 1992. Toxicological profile for antimony. Atlanta: US Department of Health and Human Services.

Sandberg A. 2002. Bioavailability of minerals in legumes. *British J of Nutrition*. 88:S281-5. Cited in National Institutes of Health, Office of Dietary Supplements. Dietary supplement fact sheet: iron. Available at: <http://dietary-supplements.info.nih.gov/factsheets/iron.asp#en1>. Last accessed 13 March 2008.

Sandström B, Abrahamson H. 1989. Zinc absorption and achlorhydria. *Eur J Clin Nutr* 43:877-879. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Sandström B, Cederblad A. 1980. Zinc absorption from composite meals: II. Influence of the main protein source. *Am J Clin Nutr* 33:1778-1783. Cited in Agency for Toxic Substances and

Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Sandström B, Sandberg AS. 1992. Inhibitory effects of isolated inositol phosphates on zinc absorption in humans. *Journal of Trace Elements and Electrolytes in Health and Disease* 6(2):99-103. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Schroeder HA, Mitchner M, Nason AP. 1970. Zirconium, niobium, antimony, vanadium and lead in rats: life term studies. *J Nutrition*. 100: 59-66. Cited in US Environmental Protection Agency. 2008. Integrated Risk Information System (IRIS). Summary for antimony. Available at: <http://www.epa.gov/ncea/iris/subst/0006.htm>. Last accessed 7 April 2008.

Schroeder WH, Dobson M, Kane DM, et al. 1987. Toxic trace elements associated with airborne particulate matter: A review. *J Air Pollut Control Assoc* 37:1267-1285. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

Schoer J. 1984. Thallium. In: Hutzinger O ed. *The handbook of environmental chemistry*. Volume 3: Anthropogenic compounds, Part C. Berlin, Springer Verlag, pp 143-214. Cited in International Programme on Chemical Safety. Thallium. Environmental health criteria. Vol. 182. World Health Organization, Geneva, Switzerland.

Sendelbach LE, Klaassen CD. 1988. Kidney synthesizes less metallothionein than liver in response to cadmium chloride and cadmium-metallothionein. *Toxicol Appl Pharmacol* 92:95-102. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

Shacklette HT and JG Boerngen. 1984. Element concentrations in soils and surficial materials of the conterminous United States. US Geological Survey Professional Paper 1270. US Government Printing Office: Washington.

Smith IC, Carson BL. 1977. Trace metals in the environment: Volume 1 - Thallium. Ann Arbor, Michigan, Ann Arbor Science Publishers, Inc. Cited in International Programme on Chemical Safety. Thallium. Environmental health criteria. Vol. 182. World Health Organization, Geneva, Switzerland.

Smith RA, Alexander RB, Wolman MG. 1987. Water-quality trends in the nation's rivers. *Science* 235:1607-1615. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services. Also cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Sommers SC, McManus RG. 1953. Multiple arsenical cancers of the skin and internal organs. *Cancer* 6:347-359. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Spencer H, Kramer L, Osis D. 1985. Zinc metabolism in man. *J Environ Pathol Toxicol Oncol* 5:265-278. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Stanek EJ and Calabrese EJ. 2000. Daily soil ingestion estimates for children at a superfund site. *Risk Anal.* 20(5):627-635.

Tam GKH, Charbonneau SM, Bryce F, et al. 1979. Metabolism of inorganic arsenic (⁷⁴As) in humans following oral ingestion. *Toxicol Appl Pharmacol* 50:319-322. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Tapiero H, Gate L, Tew KD. 2001. Iron: deficiencies and requirements. *Biomed Pharmacother.* 55:324-32. Cited in National Institutes of Health, Office of Dietary Supplements. Dietary supplement fact sheet: iron. Available at: <http://dietary-supplements.info.nih.gov/factsheets/iron.asp#en1>. Last accessed 13 March 2008.

Tay C, Seah C. 1975. Arsenic poisoning from anti-asthmatic herbal preparations. *Med J Aust* 2:424-428. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Taylor HE, Garbarino JR, Brinton TI. 1990. The occurrence and distribution of trace metals in the Mississippi River and its tributaries. *Sci Total Environ* 97/98:369-384. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

Thornton I. 1992. Sources and pathways of cadmium in the environment. *IARC Sci Publ* 118:149-162. Cited in Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium (draft for public comment). Atlanta: US Department of Health and Human Services.

Tsai M, Chien R, Hsieh S, et al. 1998. Primary hepatic angiosarcoma: Report of a case involving environmental arsenic exposure. *Chang Keng I Hsueh Tsa Chih* 21(4):469-474. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Tsai SM, Wang TN, Ko YC. 1999. Mortality for certain diseases in areas with high levels of arsenic in drinking water. *Arch Environ Health* 54(3):186-193. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Tseng, WP, Chu HM, How SW, et al. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. *J Natl Cancer Inst* 40:453-463. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Tseng, WP. 1977. Effects and dose-response relationships of cancer and Blackfoot disease with arsenic. *Environ Health Perspect* 19:109-119. Cited in Agency for Toxic Substances and Disease

Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

[URS] URS Operating Services, Inc. 2001. Preliminary assessment, Iron Mountain Mill, Superior, Mineral County, Montana. US Environmental Protection Agency Contract NO. 68-W-00-118, TDD No. 0101-0005. July 20, 2001.

[USDA] United States Department of Agriculture. 2004. Flat Creek tailings, Lolo National Forest: final site investigation. USDA Forest Service, Region 1. February 18, 2004.

Uzel C, Conrad ME. 1998. Absorption of heme iron. *Semin Hematol* 35:27-34. Cited in National Institutes of Health, Office of Dietary Supplements. Dietary supplement fact sheet: iron. Available at: <http://dietary-supplements.info.nih.gov/factsheets/iron.asp#en1>. Last accessed 13 March 2008.

Valentine JL, Reisbord LS, Kang HK, et al. 1985. Arsenic effects on population health histories. In: Mills CF, Bremner I, Chesters JK, eds. Trace elements in man and animals - TEMA 5: Proceedings of the Fifth International Symposium on Trace Elements in Man and Animals. Slough, UK: Commonwealth Agricultural Bureaux, 289-294. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

View Database. 1989. Agency for Toxic Substances and Disease Registry (ATSDR), Office of External Affairs, Exposure and Disease Registry Branch, Atlanta, GA. Cited in Agency for Toxic Substances and Disease Registry. 1992. Toxicological profile for antimony. Atlanta: US Department of Health and Human Services.

Wedepohl KH. 1991. The composition of the upper earth's crust and the natural cycles of selected metals. Metals in natural raw materials. Natural resources. In: Merian E, ed. Metals and their compounds in the environment. Occurrence, analysis, and biological relevance. New York, NY: VCH, 3-17. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Welch AH, Lico MS, Hughes JL. 1988. Arsenic in groundwater of the western United States. *Ground Water* 26(3):333-347. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Wester, R.C., H.I. Maibach, L. Sedik, J. Melendres, S. Dizio, and M. Wade. 1992. In Vitro Percutaneous Absorption of Cadmium from Water and Soil into Human Skin. *Fund. Appl. Toxicol.*, 19: 1-5. Cited in Battelle and Exponent. 2000. Final guide for incorporating bioavailability adjustments into human health and ecological risk assessments at US Navy and Marine Corps Facilities. Part 1: overview of metals bioavailability. Prepared for Naval Facilities Engineering Service Center and Engineering Field Activity West.

Wester RC, Maibach HI, Sedik L, et al. 1993. *In vivo* and *in vitro* percutaneous absorption and skin decontamination of arsenic from water and soil. *Fundam Appl Toxicol* 20(3):336-340. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

[WHO] World Health Organization. 1973. Trace elements in human nutrition: manganese. Report of a WHO expert committee. Geneva, Switzerland: Technical Report Service, 532, WHO. p. 34–36. Cited in US Environmental Protection Agency. Integrated Risk Information System (IRIS). Manganese (CASRN 7439-96-5). Available at: <http://www.epa.gov/ncea/iris/subst/0373.htm>. Last accessed 14 March 2008.

[WHO] World Health Organization. 1981. Environmental health criteria 17: Manganese. World Health Organization, Geneva, Switzerland. Cited in Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese (draft for public comment). Atlanta: US Department of Health and Human Services.

Yadrick MK, Kenney MA, Winterfelt EA. 1989. Iron, copper, and zinc status: Response to supplementation with zinc or zinc and iron in adult females. *Am J Clin Nutr* 49:145-150. Cited in Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for zinc. Atlanta: US Department of Health and Human Services.

Zaldívar R. 1974. Arsenic contamination of drinking water and foodstuffs causing endemic chronic poisoning. *Beitr Pathol* 151:384-400. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Zaldívar R, Prunés L, Ghai G. 1981. Arsenic dose in patients with cutaneous carcinomata and hepatic haemangio-endothelioma after environmental and occupational exposure. *Arch Toxicol* 47:145-154. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

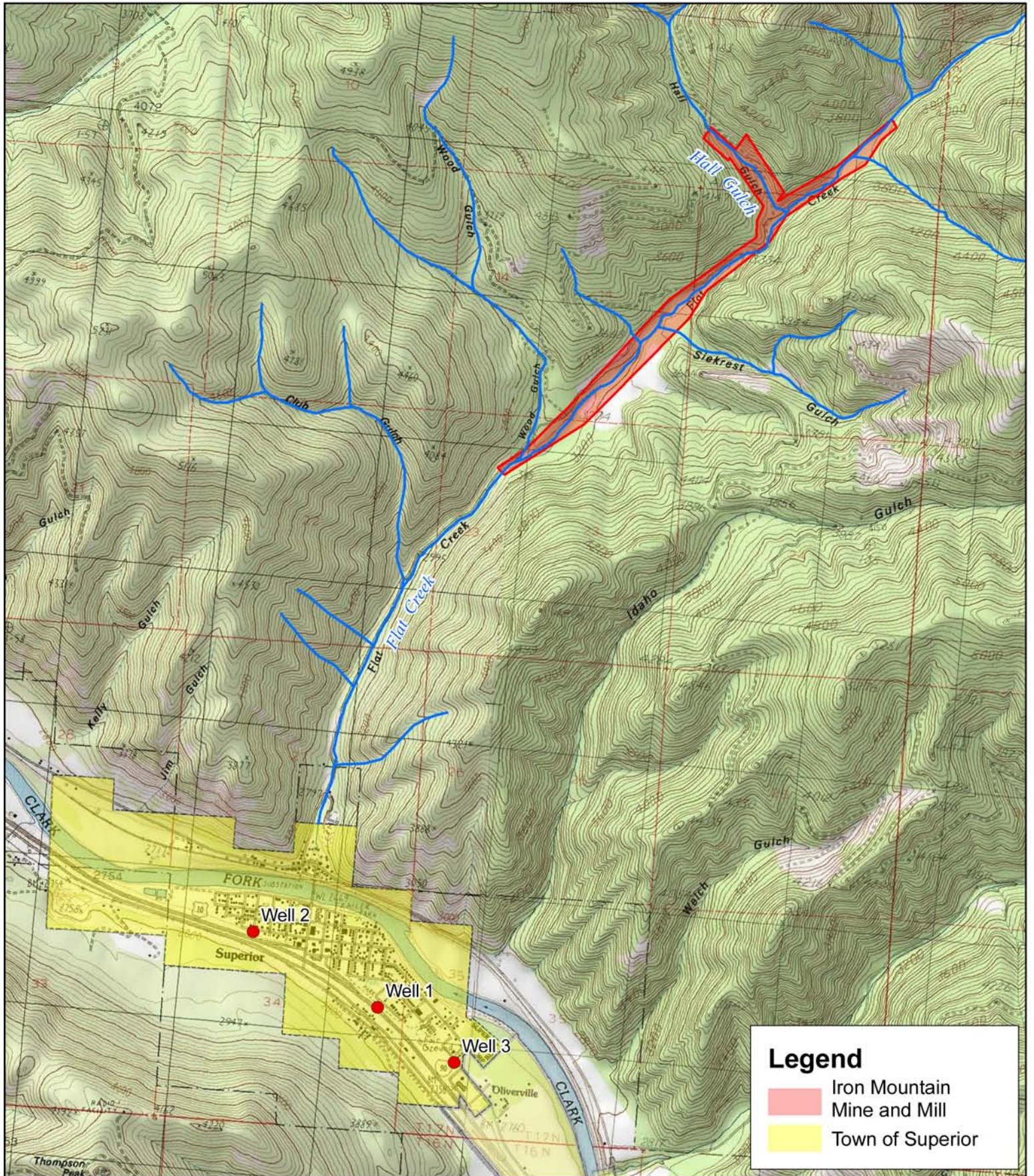
Zheng Y, Wu J, Ng JC, et al. 2002. The absorption and excretion of fluoride and arsenic in humans. *Toxicol Lett* 133(1):77-82. Cited in Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services.

Zitko V, Carson WV, Carson WG. 1975. Thallium: Occurrence in the environment and toxicity to fish. *Bull Environ Contam Toxicol* 13:23-30. Cited in Agency for Toxic Substances and Disease Registry. 1992. Toxicological profile for thallium. Atlanta: US Department of Health and Human Services.

Appendix A: Figures

Figure 1

Flat Creek IMM Site Mineral County, Montana



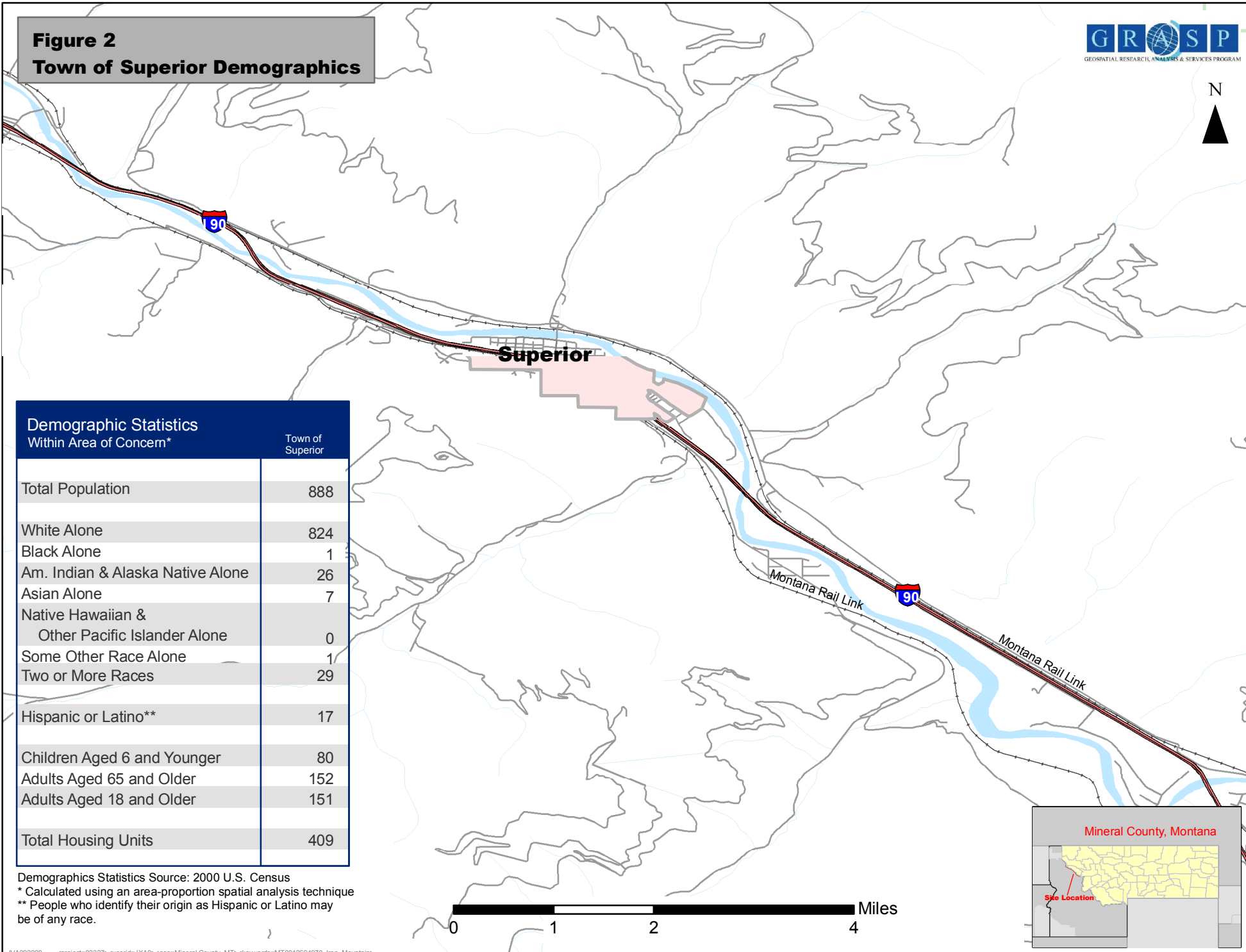
Legend

- Iron Mountain Mine and Mill
- Town of Superior

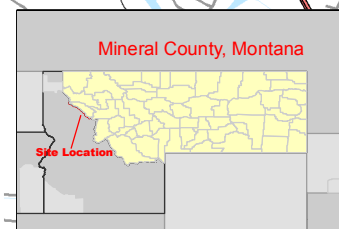




Figure 2
Town of Superior Demographics



Demographic Statistics Within Area of Concern*	Town of Superior
Total Population	888
White Alone	824
Black Alone	1
Am. Indian & Alaska Native Alone	26
Asian Alone	7
Native Hawaiian & Other Pacific Islander Alone	0
Some Other Race Alone	1
Two or More Races	29
Hispanic or Latino**	17
Children Aged 6 and Younger	80
Adults Aged 65 and Older	152
Adults Aged 18 and Older	151
Total Housing Units	409



Demographics Statistics Source: 2000 U.S. Census
 * Calculated using an area-proportion spatial analysis technique
 ** People who identify their origin as Hispanic or Latino may be of any race.

0 1 2 4 Miles

Appendix B: Tables

Table 1. Completed Exposure Pathways

Pathway Name	Completed Exposure Pathway Elements					Time Frame
	Sources	Fate and Transport	Point of Exposure	Route of Exposure	Exposed Population	
Sediment and Waste Tailings	Previous Iron Mountain Mine and Mill plant operating activities, waste tailings, mine adit discharges, and naturally occurring mineral deposits	Waste tailings piles on the mine site and those that erode into Flat Creek and distribute throughout the floodplain area	Mine site and Flat Creek floodplain area as it passes through the mine site and continues toward Superior	Incidental Ingestion, Dermal contact	People engaged in hunting, hiking, wading, fishing and other recreational activities	Past, Present, Future
Soil	Waste tailings and naturally occurring mineral deposits	Waste tailings erode into soil and some were transported by residents to be used as fill material throughout Superior	Mine site and the Flat Creek floodplain area, residences, public areas, and rights-of-way in Superior	Incidental Ingestion, Dermal contact	People engaged in recreational activities, Town of Superior residents and area visitors	Past, Present, Future
Dust	Waste tailings, and sediment and soil affected by the tailings	Waste tailings that are disturbed and re-suspended in the air	Mine site, Flat Creek Floodplain and the town of Superior	Incidental Ingestion, Dermal contact	People engaged in recreational activities, Town of Superior residents and area visitors	Past, Present, Future
Surface Water	Waste tailings and naturally occurring mineral deposits	Contaminants in waste tailings that leach into Flat Creek surface water	Flat Creek floodplain area as it passes through the mine site and continues toward Superior	Incidental Ingestion, Dermal contact	People engaged in wading, fishing and other recreational activities	Past, Present, Future
			Tap water from Flat Creek	Ingestion, Dermal contact	People with a water intake on Flat Creek	Past, Present, Future
Groundwater	Waste tailings, mine adit discharges, and naturally occurring mineral deposits	Contaminants in waste tailings potentially leach into shallow groundwater; Flat Creek and Hall Gulch surface water potentially impacts shallow groundwater	Tap water from Flat Creek Spring	Ingestion, Dermal contact	Town of Superior residents	Past
			Tap water from private wells	Ingestion, Dermal contact	Town of Superior residents	Past, Present, Future
			Tap water from Town of Superior Wells	Ingestion, Dermal contact	Town of Superior residents	Past, Present, Future

Table 2. Potential and Eliminated Exposure Pathways

Pathway Name	Potential Exposure Pathway Elements					Time Frame
	Sources	Fate and Transport	Point of Exposure	Route of Exposure	Exposed Population	
Groundwater	Waste tailings, mine adit discharges, and naturally occurring mineral deposits	Waste tailings potentially leach into shallow groundwater; Flat Creek and Hall Gulch surface water potentially impacts shallow groundwater	Tap water from Flat Creek Spring	Ingestion, Dermal contact	Town of Superior residents, if the spring is used as an emergency supply	Future
Pathway Name	Eliminated Exposure Pathway Elements					Time Frame
	Sources	Fate and Transport	Point of Exposure	Route of Exposure	Exposed Population	
Surface Water	Mine adit discharges	Mine adit discharges distribute in the Hall Gulch area	Hall Gulch area	None	None	Present, Future
Groundwater	Waste tailings, mine adit discharges, and naturally occurring mineral deposits	Waste tailings potentially leach into shallow groundwater; Flat Creek and Hall Gulch surface water potentially impacts shallow groundwater	Tap water from Flat Creek Spring	None	None	Present

Table 3. Concentrations of Metals in Iron Mountain and Mill Site and Flat Creek Floodplain Area Sediments

<i>Analyte*</i>	<i>Frequency of Detection†</i>	<i>Minimum Detected Concentration (ppm)</i>	<i>Average Concentration (ppm)</i>	<i>Maximum Detection Concentration (ppm)</i>	<i>Location of Maximum Detected Concentration</i>	<i>Comparison Value (ppm)</i>	<i>Frequency Above Comparison Value‡</i>
Antimony	13/13	30.9	382	1,910	Near mine adit	20 (RMEG-child)	13/13
Arsenic	13/13	71.4	3,340	40,200	Near mine adit	0.5 (CREG) 20 (EMEG-child)	13/13 13/13
Cadmium	13/13	3.3	20	59.7	Lower USDA Forest Service/ ARSARCO Boundary	10 (EMEG-child)	7/13
Iron	6/6	12,400	64,700	298,000	Near mine adit	23,000 (RBC residential)	2/6
Lead	13/13	115	2,500	9,470	Lower USDA Forest Service/ ARSARCO Boundary	400 (SSL)	12/13
Manganese	13/13	486	6,180	66,100	Near mine adit	3,000 (RMEG-child)	2/13
Thallium	1/6	--	--	11.5 J	Near mine adit	5.1 (RBC residential)	1/6
Zinc	13/13	654	9,450	75,900	Near mine adit	20,000 (EMEG-child)	1/13

References: EPA 2002a, USDA 2004

* Only analytes detected above health-based comparison values are provided in the table.

† Frequency of detection is a ratio of the number of times an analyte was detected to the total number of samples collected during the sampling event.

‡ Frequency above comparison values is a ratio of the number of times the analyte was detected above the comparison value to the total number of samples collected during the sampling event.

CREG cancer risk evaluation guide

EMEG environmental media evaluation guide for chronic exposure durations

J estimated value between the detection limit and the limit of quantitation (considered a valid sample)

ppm parts per million

RMEG reference dose media evaluation guide

RBC risk-based concentration

SSL soil screening level

-- not applicable

Table 4. Concentrations of Metals in Iron Mountain and Mill Site and Flat Creek Floodplain Area Waste Tailings

<i>Analyte*</i>	<i>Frequency of Detection[†]</i>	<i>Minimum Detected Concentration (ppm)</i>	<i>Average Concentration (ppm)</i>	<i>Maximum Detection Concentration (ppm)</i>	<i>Location of Maximum Detected Concentration</i>	<i>Comparison Value (ppm)</i>	<i>Frequency Above Comparison Value[‡]</i>
Antimony	14/14	13.4	1,930	4,500	Near Mill Foundation along Flat Creek	20 (RMEG-child)	14/14
Arsenic	14/14	33.7	2,510	9,350	Along Flat Creek	0.5 (CREG) 20 (EMEG-child)	14/14 14/14
Cadmium	14/14	1.3	58	161	Along Flat Creek near Private Residence	10 (EMEG-child)	9/14
Iron	9/9	13,600	71,100	83,100	Mill Foundation Slope	23,000 (RBC residential)	5/9
Lead	14/14	92.2	15,400	55,600	Along Flat Creek near Private Residence	400 (SSL)	13/14
Manganese	14/14	469	2,920	5,900	Downstream of Lower USDA Forest Service/ ARSARCO Boundary	3,000 (RMEG-child)	7/14
Thallium	6/9	2.6	8	12.6	Along Flat Creek near Private Residence	5.1 (RBC residential)	5/9
Zinc	14/14	677	10,000	25,500	Test Pit #3	20,000 (EMEG-child)	4/14

References: EPA 2002a, USDA 2004

* Only analytes detected above health-based comparison values are provided in the table.

† Frequency of detection is a ratio of the number of times an analyte was detected to the total number of samples collected during the sampling event.

‡ Frequency above comparison values is a ratio of the number of times the analyte was detected above the comparison value to the total number of samples collected during the sampling event.

CREG cancer risk evaluation guide

EMEG environmental media evaluation guide for chronic exposure durations

J estimated value between the detection limit and the limit of quantitation (considered a valid sample)

ppm parts per million

RMEG reference dose media evaluation guide

RBC risk-based concentration

SSL soil screening level

-- not applicable

Table 5. Arsenic and Lead Concentrations in Town of Superior Soil — Field Screening Results

<i>Town of Superior Sampling Location</i>	<i>Analyte</i>	<i>Frequency of Detection*</i>	<i>Minimum Detected Concentration (ppm)</i>	<i>Maximum Detected Concentration (ppm)</i>	<i>Comparison Value (ppm)</i>	<i>Frequency Above Comparison Value[†]</i>
Rights-of-Way	Arsenic	70/381	23 J	400	0.5 (CREG) 20 (EMEG-child)	70/381 70/381
	Lead	253/381	19 J	2,200	400 (SSL)	7/381
Residential	Arsenic	14/158	43 J	1,400	0.5 (CREG) 20 (EMEG-child)	14/158 14/158
	Lead	106/158	17 J	9,400	400 (SSL)	11/158
Town/Open Space	Arsenic	6/58	47 J	1,500	0.5 (CREG) 20 (EMEG-child)	6/58 6/58
	Lead	36/58	21 J	7,700	400 (SSL)	5/58
High School Track	Arsenic	3/11	370	1,800	0.5 (CREG) 20 (EMEG-child)	3/11 3/11
	Lead	7/11	35 J	9,200	400 (SSL)	4/11

Reference: EPA 2002a

* Frequency of detection is a ratio of the number of times an analyte was detected to the total number of samples collected during the sampling event.

† Frequency above comparison value is a ratio of the number of times the analyte was detected above comparison values to the total number of samples collected during the sampling event.

CREG cancer risk evaluation guide

EMEG environmental media evaluation guide for chronic exposure durations

J estimated value between the detection limit and the limit of quantitation (considered a valid sample)

ppm parts per million

SSL soil screening level

Table 6. Arsenic and Lead Concentrations in Town of Superior Soil — Laboratory Results

<i>Town of Superior Sampling Location</i>	<i>Analyte</i>	<i>Frequency of Detection*</i>	<i>Minimum Detected Concentration (ppm)</i>	<i>Average Concentration (ppm)</i>	<i>Maximum Detected Concentration (ppm)</i>	<i>Comparison Value (ppm)</i>	<i>Frequency Above Comparison Value[†]</i>
Rights-of-Way	Arsenic	23/24	2.7	73	582	0.5 (CREG) 20 (EMEG-child)	23/24 9/24
	Lead	26/26	0.38	405	2,320	400 (SSL)	7/26
Residential	Arsenic	29/29	3.6	197	2,620	0.5 (CREG) 20 (EMEG-child)	29/29 11/29
	Lead	27/27	12.4	1,230	16,700	400 (SSL)	10/27
Town/Open Space	Arsenic	6/6	4.6	503	1,910	0.5 (CREG) 20 (EMEG-child)	6/6 5/6
	Lead	6/6	11.3	2,601	9,360	400 (SSL)	5/6
High School Track	Arsenic	4/5	250	904	2,110	0.5 (CREG) 20 (EMEG-child)	4/5 4/5
	Lead	5/5	2.4	3,806	10,700	400 (SSL)	4/5

Reference: EPA 2002a

* Frequency of detection is a ratio of the number of times an analyte was detected to the total number of samples collected during the sampling event.

† Frequency above comparison values is a ratio of the number of times the analyte was detected above comparison values to the total number of samples collected during the sampling event.

CREG cancer risk evaluation guide

EMEG environmental media evaluation guide for chronic exposure durations

J estimated value between the detection limit and the limit of quantitation (considered a valid sample)

ppm parts per million

SSL soil screening level

Table 7. Maximum Concentrations of Metals in Town of Superior Soils — Field Screening and Laboratory Results

<i>Analyte*</i>	<i>Field Screening Maximum Detected Concentration (ppm)</i>	<i>Laboratory Maximum Detected Concentration (ppm)</i>	<i>Comparison Value (ppm)</i>
Antimony	2,500	2,360	20 (RMEG-child)
Cadmium	220 J	46.3	10 (EMEG-child)
Iron	53,000	64,800	23,000 (RBC residential)
Manganese	3,900	4,250	3,000 (RMEG-child)
Thallium	--	6.4	5.1 (RBC residential)

Reference: EPA 2002a

* Only analytes detected above health-based comparison values are provided in the table.

EMEG environmental media evaluation guide for chronic exposure durations

J estimated value between the detection limit and the limit of quantitation (considered a valid sample)

ppm parts per million

RBC risk-based concentration

RMEG reference dose media evaluation guide

-- not applicable

Table 8: Concentrations of Metals in Surface Water

<i>Location</i>	<i>Analyte*</i>	<i>Frequency of Detection[†]</i>	<i>Minimum Detected Concentration (ppb)</i>	<i>Average Concentration (ppb)</i>	<i>Maximum Detection Concentration (ppb)</i>	<i>Comparison Value (ppb)</i>	<i>Frequency Above Comparison Value[‡]</i>
Hall Gulch/ Mine Adit	Antimony	5/5	11	123	472	4 (RMEG-child) 6 (MCL)	5/5 5/5
	Arsenic	13/13	15	852	4,150	0.02 (CREG) 3 (EMEG-child) 10 (MCL)	13/13 13/13 13/13
	Cadmium	6/13	0.3	4.08	21.8	2 (EMEG-child)	1/13
	Iron	9/9	11	7,770	50,400	NA	–
	Lead	8/13	4	262	1,990	15 (MCL)	4/13
	Manganese	8/9	14	6,690	27,100	500 (RMEG-child)	7/9
	Zinc	13/13	19	7,850	58,100	3,000 (EMEG-child)	7/13
Flat Creek	Antimony	23/24	7.2	21.6	34.6	04 (RMEG-child) 6 (MCL)	23/24 23/24
	Arsenic	10/24	3.8	4.7	6	0.02 (CREG) 3 (EMEG-child) 10 (MCL)	10/24 10/24 0/24
	Cadmium	9/24	0.4	1.22	1.6	2 (EMEG-child)	0/24
	Iron	1/5	–	–	96.5	NA	–
	Lead	20/24	3.3	23.9	233	15 (MCL)	7/24
	Manganese	11/17	2.8	7.22	23.5	500 (RMEG-child)	0/17
	Zinc	24/24	10.9	161	318	3,000 (EMEG-child)	0/24

References: EPA 2002a, MFG 2000, USDA 2004

* Only analytes detected above health-based comparison values in either Hall Gulch or Flat Creek surface water are provided in the table.

† Frequency of detection is a ratio of the number of times an analyte was detected to the total number of samples collected during the sampling event.

‡ Frequency above comparison values is a ratio of the number of times the analyte was detected above the comparison value to the total number of samples collected during the sampling event.

CREG cancer risk evaluation guide

EMEG environmental media evaluation guide for chronic exposure durations

J estimated value between the detection limit and the limit of quantitation (considered a valid sample)

MCL maximum contaminant level for drinking water

Flat Creek IMM

NA none available
ppb parts per billion
RMEG reference dose media evaluation guide
-- not applicable

Table 9: Concentrations of Metals in Groundwater

<i>Sampling Location</i>	<i>Analyte*</i>	<i>Frequency of Detection[†]</i>	<i>Minimum Detected Concentration (ppb)</i>	<i>Maximum Detected Concentration (ppb)</i>	<i>Comparison Value (ppb)</i>	<i>Frequency Above Comparison Value[‡]</i>
Distribution System	Antimony	10/43	1	30	4 (RMEG-child) 6 (MCL)	1/43 1/43
	Arsenic	1/3	ND	1	0.02 (CREG) 3 (EMEG-child) 10 (MCL)	1/3 0/3 0/3
	Cadmium	1/3	ND	3	2 (EMEG-child) 5 (MCL)	1/3 0/3
	Lead	10/13	1	10	15 (MCL)	0/13
Wells 1, 2, and 3	Antimony	3/38	2	10	4 (RMEG-child) 6 (MCL)	1/38 1/38
	Arsenic	6/26	1	4	0.02 (CREG) 3 (EMEG-child) 10 (MCL)	6/26 1/26 0/26
	Lead	0/7	ND	ND	15 (MCL)	0/7
Flat Creek Spring	Antimony	20/33	2	34	4 (RMEG-child) 6 (MCL)	19/33 19/33
	Arsenic	3/7	1	1	0.02 (CREG) 3 (EMEG-child) 10 (MCL)	3/7 0/7 0/7
	Lead	0/1	ND	ND	15 (MCL)	0/1

References: EPA 2002a, MDEQ 2000, MDEQ 2007

* Only analytes detected above health-based comparison values are provided in the table, with the exception of lead. Lead is included in the table because of its elevated detection in other media (soil and surface water).

† Frequency of detection is a ratio of the number of times an analyte was detected to the total number of samples collected during the sampling event.

‡ Frequency above comparison values is a ratio of the number of times the analyte was detected above the comparison value to the total number of samples collected during the sampling event.

CREG cancer risk evaluation guide

EMEG environmental media evaluation guide for chronic exposure durations

J estimated value between the detection limit and the limit of quantitation (considered a valid sample)

MCL maximum contaminant level for drinking water

ppb parts per billion

Flat Creek IMM

RMEG reference dose media evaluation guide

Table 10. Blood Lead Results for Town of Superior Residents

<i>Age Group</i>	<i>Number of Residents Tested</i>	<i>Range of Blood Lead Concentrations (µg/dL)</i>
0 to 7 years	8 children	<1 – 2.6
8 to 13 years	6 children	<1 – 2.0
14 to 18 years	24 people	<1 – 3.5
19 to 75 years	23 people	<1 – 6.9
Age not available	5 people	<1 – 2.1

Reference: MCEPH 2002d

µg/dL micrograms per deciliter

Appendix C: Exposure Dose Equations

Exposure Dose Equations

Estimating an exposure dose requires identifying how much, how often, and how long a person may come in contact with some concentration of the chemical in a specific medium. The equations and assumptions used to estimate exposure doses follow.

Water Exposures

Exhibit 1: Exposure Dose Equation for Ingestion of Water

$$D = \frac{C \times IR \times EF \times AF}{BW}$$

where,

D	=	exposure dose in milligrams per kilogram per day (mg/kg/day)
C	=	chemical concentration in milligrams per liter (mg/L)
IR	=	intake rate in liters per day (L/day)
EF	=	exposure factor (unitless)
AF	=	bioavailability factor
BW	=	body weight in kilograms (kg)

In the absence of complete exposure-specific information, ATSDR applied several conservative exposure assumptions to define site-specific exposures as accurately as possible. Specifically, ATSDR estimated exposure doses using the following general assumptions and default intake rates for exposure through ingestion of water:

- The intake rate for drinking water for a child was assumed to be 1 L/day and an adult was assumed to be 2 L/day.
- The intake rate for incidental ingestion of water while wading or fishing for both children and adults was assumed to be 0.15 L/day, representing 3 hours of wading or fishing per day with an intake rate of 50 milliliters per hour (mL/hour).
- The exposure factor was assumed to be 1, representing daily exposure.
- The body weight of a child was assumed to be
 - 10 kg, when representing daily drinking water exposure, and
 - 16 kg, when representing wading or fishing in Flat Creek.
- The body weight of an adult was assumed to be 70 kg.
- The bioavailability was assumed to be 100%, unless otherwise indicated in the main text of this document.

Soil Exposures

Exhibit 2: Exposure Dose Equation for Ingestion of Soil

$$D = \frac{C \times IR \times EF \times AF \times CF}{BW}$$

where,

D	=	exposure dose in milligrams per kilogram per day (mg/kg/day)
C	=	chemical concentration in milligrams per kilogram (mg/kg)
IR	=	intake rate in milligrams per day (mg/day)
EF	=	exposure factor (unitless)
AF	=	bioavailability factor
CF	=	conversion factor, 1×10^{-6} kilograms/milligram (kg/mg)
BW	=	body weight in kilograms (kg)

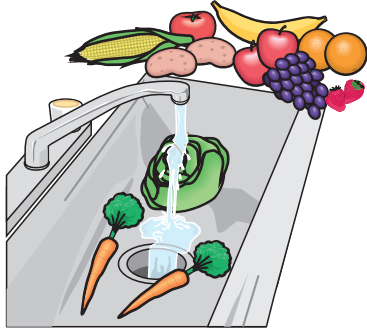
In the absence of complete exposure-specific information regarding soil exposures, ATSDR applied several conservative exposure assumptions to define site-specific exposures as accurately as possible. Specifically, ATSDR estimated exposure doses using the following general assumptions and default intake rates for exposure through ingestion of soil:

- The intake rate for a child was assumed to be 200 mg/day and an adult was assumed to be 100 mg/day.
- The exposure factor was assumed to be 1, representing daily exposure.
- The body weight of a child was assumed to be 16 kg and an adult was assumed to be 70 kg.
- The bioavailability was assumed to be 100%, unless otherwise indicated in the main text of this document.

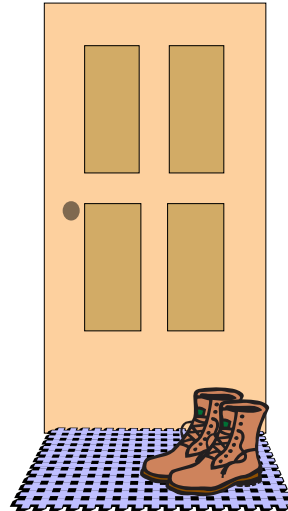
Appendix D: Ways to protect your health

Ways to protect your health

By keeping dirt from getting into your house and into your body



Wash and peel all fruits, vegetables, and root crops



Wipe shoes on doormat or remove shoes



Don't eat food, chew gum, or smoke when working in the yard



Damp mop floors and damp dust counters and furniture regularly



Wash dogs regularly



Wash children's toys regularly



Wash children's hands and feet after they have been playing outside